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ETIOLOGY, DIAGNOSIS AND TREATMENT OF THYROID FAILURE

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THYROID failure may be defined as the failure of the thyroid gland to secrete an adequate amount of hormone necessary to maintain a normal rate of body metabolism, thereby resulting in a characteristic group of symptoms and signs. The terms hypothyroidism and myxedema have been defined respectively to indicate mild and severe thyroid failure, but here they shall be used synonymously. The purpose of this paper is to present a review of our experience with the causation, differential diagnosis and treatment of thyroid failure.

The mildest degree of thyroid failure is asymptomatic and may be characterized only by a lowered basal metabolic rate. Actually, this stage is rarely diagnosed and in our experience is usually seen in those patients who have gradual enlargement of the thyroid gland due to struma lymphomatosa (Hashimoto's struma). Treatment with thyroid extract will elevate the basal metabolic rate 20 to 30 per cent, but clinically these patients may feel just the same. It is probably not of great importance to diagnose the disease at this stage.

The early stage of symptomatic thyroid failure is characterized by cold intolerance, dryness of the skin and fatigue, as well as a low basal metabolic rate, but there are no symptoms or signs of the deposition of myxedematous fluid in the intercellular tissues. The patients in this stage of the disease are difficult to diagnose and are equally difficult to separate from that large group of patients with normal thyroid function who have similar symptoms.

Most important is the diagnosis of advanced thyroid failure which disables the patient. Not only are the mild symptoms of hypothyroidism present but, in addition, all the symptoms and signs due to the accumulation of myxedematous fluid. These will be described later.

ETIOLOGY

Hypothyroidism may be due to intrinsic disease of the thyroid or extrinsic causes (Table 1).

Hypothyroidism as a result of extrinsic causes is probably more common than that due to intrinsic disease. However, the latter often contributes to so-called extrinsic hypothyroidism as will be shown.

Total thyroidectomy for any reason whatsoever could be expected to cause thyroid failure. Actually, most surgeons will agree that it is difficult to do a total thyroidectomy. In our own experience myxedema is seen after total thyroidectomy performed for alveolar or papillary carcinoma involving both lobes.

Table 1

Causes of Thyroid Failure

Extrinsic

1. Surgical
   a. Total thyroidectomy
   b. Partial thyroidectomy (for Graves' disease, struma lymphomatosa)
2. Radiation therapy
   a. Radioactive iodine (Graves' disease)
   b. X-ray therapy (struma lymphomatosa)
3. Antithyroid drugs
   a. Thiouracil derivatives
   b. Thiocyanates

Intrinsic

1. Chronic thyroiditis
   a. Struma lymphomatosa (Hashimoto)
   b. Struma fibrosa (Riedel)
2. Spontaneous atrophy
   a. Primary
   b. Secondary to anterior pituitary failure
3. Congenital
   a. Atrophy
   b. Iodine deficient diet

Partial thyroidectomy per se does not usually result in immediate postoperative hypothyroidism unless the patient has Hashimoto's struma or hyperplastic goiter of Graves' disease extensively infiltrated with a fibrolymphocytic type of thyroiditis.

Hypothyroidism has been frequently observed in our experience in patients who have had a partial thyroidectomy for Hashimoto's goiter. The surgery definitely accelerates this development. Therefore, we believe all such patients should be routinely placed on permanent thyroid medication postoperatively even if they present no evidence of thyroid failure at the time of surgery.

Whitesell and Black[1] in a pathologic study of 86 resected hyperplastic thyroid glands of Graves' disease with lymphocytic and fibrolymphocytic replacement noted that the incidence of postoperative myxedema was directly proportional to the degree of replacement of the thyroid tissue. In patients whose partially resected thyroid glands showed 40 to 50 per cent replacement or more, the incidence of postoperative myxedema was 70 per cent according to these authors. Therefore, we believe postoperative thyroid medication should also be prescribed routinely in this group of patients. In our experience most patients who are found to have myxedema have undergone partial thyroidectomy for Graves' disease anywhere from 2 months to 30 years earlier.

Thyroid failure rarely occurs after partial thyroidectomy for toxic or nontoxic adenomatous goiter.

The increasingly widespread use of radioactive iodine especially in treating Graves' disease is making this agent an important cause of thyroid failure. The incidence of myxedema has been about 10 per cent of all patients with Graves' disease who were so treated. However, a small number of these patients have been found to have only temporary myxedema. External roentgen therapy to the thyroid gland usually will not cause thyroid failure unless the patient has Hashimoto's goiter.

Two important causes of reversible thyroid failure are the thiouracil and thiocyanate drugs. The use of thiouracil derivatives, especially in treating Graves' disease, may result in an occasional case of myxedema which will disappear when the drug is withdrawn. A less well recognized cause of myxedema is the thiocyanate group of drugs used especially in the treatment of hypertension. The myxedema may or may not be accompanied by thyroid enlargement. Both will disappear if the thiocyanate is stopped without using thyroid extract.

In cases of thyroid failure attributable to intrinsic disease it is becoming increasingly apparent to us that most spontaneous thyroid failure is probably due to a lymphocytic infiltration into the thyroid gland with secondary atrophy of the thyroid secretory cells. This type of thyroiditis may or may not be different from Hashimoto's struma. The use of the Vim-Silverman needle for biopsy of the thyroid gland has led us to earlier diagnoses of lymphocytic thyroiditis whether in the presence or absence of thyroid failure. When the diagnosis is confirmed by biopsy, the patient is given thyroid extract permanently as prophylactic therapy for the development of hypothyroidism. If the thyroid is enlarged it often shrinks considerably when 2 or 3 gr. of U.S.P. thyroid extract per day is administered.[2]

Since Riedel's type of thyroiditis is so rare in contrast to Hashimoto's thyroiditis, it is not an important cause of thyroid failure.

Spontaneous atrophy of the thyroid gland with fibrosis and no lymphocytic infiltration is seldom observed as a primary process but is more usually secondary to failure of the anterior lobe of the pituitary gland. The degree of secondary thyroid failure depends upon the extent of the failure of the anterior pituitary gland. Severe thyroid failure is rare in patients with anterior pituitary failure.

Cretinism in this country is usually due to intra-uterine athyreosis or atrophy of the thyroid of the fetus. In other countries, such as Switzerland, it may be a result of iodine deficiency in the diet and, furthermore, is characterized by the presence of a goiter.

DIAGNOSIS

Symptoms and Signs

It is well known that the onset of the symptoms and signs of thyroid failure is usually so insidious that many patients will not describe many of the classic

symptoms when the original history is recorded. If the condition is suspected, direct questioning will usually elicit most of the classic symptoms. For this reason it has been our experience that the history is of less value than the physical findings. The important symptoms and signs of thyroid failure as we have encountered them are summarized as follows:

Cold Tolerance. This is a symptom which is difficult to evaluate because many euthyroid patients especially women are sensitive to cold. However, the latter have a long history of cold intolerance while that of the hypothyroid patient is usually of recent onset. The patient should be carefully questioned about cold intolerance. For example: When outside, do you wear more clothes than formerly? Were you comfortable in the extreme heat of the summer while everyone else sweltered? and finally, Do you want the temperature of the house unreasonably hot and do you need more covers on your bed at night than formerly? Occasionally, we have seen a patient with obvious myxedema who denies ever having any cold intolerance.

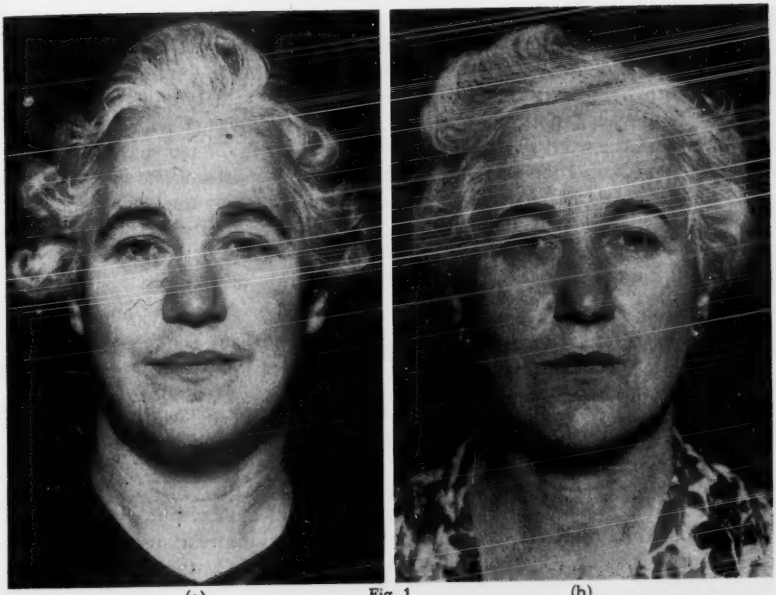
Fatigue. Unfortunately this symptom is not too indicative of thyroid failure since it is nonspecific and present in most other organic diseases as well as functional syndromes.

Dryness of Skin. This symptom and sign must be carefully evaluated before it will be of much help in the diagnosis of the hypothyroid patient. First, the dryness of the skin must have begun fairly recently. Second, the dryness of the skin is general. The degree of dryness may vary, but most patients with myxedema have a "sandpaper-like" skin especially evident over the back and often with excessive flaking. It must be kept in mind that there are other causes of dry skin such as sensitivity to soap and detergents which may be localized to the forearms and hands. Since dryness and loss of hair are not limited to the hypothyroid patient, they are of little aid to the diagnosis. Nail changes are not very helpful either.

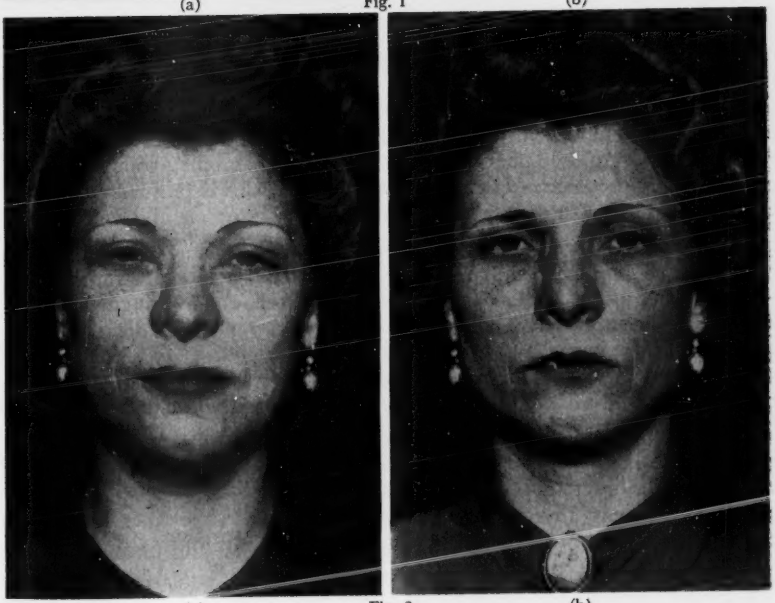
Myxedema Facies. The characteristic facies of the myxedematous patient has been the outstanding diagnostic aid in our experience. Once the characteristics of this facies are mentally recorded the diagnosis will almost invariably be made by the alert clinician before the history is taken.

This type of facies is characterized by a pale yellowish-tinged color, puffiness of the entire face, especially of the upper eyelids and portion of the cheeks above the angle of the jaw, and usually an apathetic or listless expression, well-illustrated by lusterless eyes. The lips and the tongue are thickened and coarse. Figures 1 and 2 illustrate the facies of myxedematous patients before and after treatment with thyroid extract.

Mental and Physical Lethargy. The patient will usually be aware of a definite slowing of mental and physical activities. Excessive drowsiness may well be present. The patient will often volunteer that he or she takes twice as long to get any work done either on the job or around the house. The speech is often slowed, husky, and slurred, occasionally simulating that of an inebriated person.



(a) Fig. 1 (b)



(a) Fig. 2 (b)

Fig. 1 and 2. (a) Facies of myxedematous patient before treatment with thyroid extract. (b) Same patient after treatment with thyroid extract.

The most specific sign of thyroid failure is the slowing of the tendon reflex responses, particularly those of the biceps and Achilles tendons. Characteristically, both phases of this reflex response are delayed, but the delay in the duration of the relaxation phase is most obvious. Lambert, Underdahl, Beckett and Mederos[3] reported that the reflex was present in 77 per cent of patients with myxedema and its incidence was directly proportional to the decrease in the basal metabolic rate. This group also showed that the slow response of the tendon reflex in myxedema was due to an abnormality of the contractile mechanism of the muscle rather than due to abnormal activity of the neural elements of the reflex or the mechanism of excitation of the muscle.

Laboratory Tests

Laboratory tests are not only important to document obvious cases of thyroid failure, but also to help differentiate the hypothyroid from the euthyroid patient. It is our opinion that the basal metabolic rate and plasma cholesterol remain the most useful tests for the diagnosis of hypothyroidism. The protein bound iodine is a good test for diagnosis of thyroid failure, but the procedure itself has certain limitations which preclude its widespread use. The radioactive iodine tracer has certain limitations in its interpretation in patients with thyroid failure.

The basal metabolic rate when properly done and interpreted is still the most satisfactory test available for the diagnosis of thyroid failure because of its ready availability and low cost. In an excellent recent review Kyle[4] discussed the pitfalls encountered in the determination and interpretation of this test. In our patients with thyroid failure the basal metabolic rate usually varies from —20 to —40 per cent. Occasionally, the basal metabolic rate will be normal in the presence of obvious thyroid failure. This is probably due to an extra thyroid factor or disease which tends to elevate the metabolic rate.

Many patients with normal thyroid function have low basal metabolic rates. The fact that most euthyroid patients have a basal metabolic rate between —15 and +15 per cent should not obscure the fact that many patients normally have basal metabolic rates above or below this range, as determined by Boothby.[5] Therefore, patients with low basal metabolic rates are not necessarily hypothyroid and should be carefully differentiated from patients with true hypothyroidism before prescribing thyroid extract.

Just as important as the initial determination of the basal metabolic rate is the repeat basal metabolic rate done after a two months therapeutic trial with thyroid extract. If the basal metabolic rate is elevated 20 or more per cent by treatment, thyroid failure is almost certain. If there is little or no change, the patient probably does not have hypothyroidism.

The plasma cholesterol is also a helpful, readily available, inexpensive aid to the diagnosis of thyroid failure. In almost all patients with thyroid failure the plasma cholesterol will be above 250 mg. per cent. Occasionally,

a value lower than this will be seen in a patient who has proved thyroid failure. One of our patients recently had a plasma cholesterol of 220 mg. per cent which dropped to 110 mg. per cent after treatment with thyroid extract. Another one of our patients with proved myxedema had a plasma cholesterol of 164 mg. per cent initially. In this case a chronic mastoiditis was responsible for the low value; low cholesterol levels are sometimes found with other infections.

It is therefore important not only to determine the initial plasma cholesterol, but also to determine the degree of fall after two months treatment with thyroid extract. As a rule the cholesterol should fall more than 100 mg. per cent if thyroid failure is present. Occasionally an initial normal cholesterol in a patient with myxedema will show little or no decrease after treatment despite the remission of the clinical signs and symptoms and definite elevation of the basal metabolic rate. The reason for this is not known and the plasma cholesterol as an aid to diagnosis is limited in cases such as these. One of our patients with myxedema had a blood cholesterol of 229 mg. per cent and a basal metabolic rate of —30 per cent. Treatment with thyroid extract resulted in an elevation of the rate to —5 per cent, but the plasma cholesterol showed only a small decrease, being 186 mg. per cent.

The protein bound iodine is the most specific test available for evaluating thyroid function since it measures protein bound organic iodine of the thyroid hormone in the circulating blood. When it is correctly performed and carefully interpreted it may be of great help. Unfortunately, it remains an extremely difficult test to perform, consequently expensive and not readily available. Furthermore, the administration of any x-ray contrast media containing organic iodine prior to the determination will result in falsely elevated values. Starr et al.[6] in a study of 39 untreated patients with myxedema found that 32 of these patients had initial protein bound iodine values below 3 micrograms per cent, generally regarded as the lowest limit of normal thyroid function. Perhaps in the future an easier method of performing the determination may be devised, making this test of much more help to the physician in private practice.

The radioactive iodine tracer test is also of limited value in the diagnosis of thyroid failure. It is a simple test to do, but the facilities are not readily available. We have found the normal range of radioactive iodine uptake in 24 hours as measured over the thyroid is 10 to 60 per cent when 100 microcuries is given. A radioactive iodine tracer uptake of less than 10 per cent is usually compatible with thyroid failure in the absence of known iodine blocking agents. However, uptakes as high as 30 per cent in 24 hours may be seen in obviously hypothyroid patients. Therefore, a normal radioactive iodine uptake does not rule out thyroid failure.

The electrocardiogram may be of some help in patients with severe myxedema by showing generalized low voltage changes especially in the T-waves. However, in many patients, the electrocardiogram may be within normal limits, and therefore the test has not been of too much help to us.

Differential Diagnosis

The important conditions to be differentiated from primary thyroid failure are hypometabolism without hypothyroidism, anterior pituitary failure, and nephrosis due to any cause.

The typical patient with hypometabolism without hypothyroidism is usually a nervous woman with or without some intolerance to cold, dryness of the skin, and fatigue, who has a lowered basal metabolic rate varying from —10 to —30 per cent. No characteristic findings of myxedema are present and the plasma cholesterol is almost always normal. The radioactive iodine tracer uptake and protein bound iodine determination are within normal range. A two month therapeutic trial with 2 gr. of U.S.P. thyroid extract will not produce any significant elevation of the basal metabolic rate or any decrease in the plasma cholesterol in such a patient, despite the observation that the patient may feel better due to the nonspecific effect of the treatment.

Most patients with anterior pituitary failure do not show any clinical signs or symptoms of thyroid failure. This is true because the degree of anterior pituitary failure may be mild to moderate. Furthermore, if the anterior pituitary deficiency is moderate to severe the thyroid gland may function independently at a lowered level sufficient to prevent most of the symptoms or signs of thyroid failure from appearing.

Younghusband, Horrax, Hurxthal, Hare and Poppen[7] have noted that in 86 patients with anterior pituitary failure due to tumor the average basal metabolic rate was —20 per cent and 27 of these 86 patients had a basal metabolic rate above —10 per cent. These authors also noted that the average plasma cholesterol was 230 mg. per cent which is slightly above the normal average.

On rare occasions a patient who has anterior pituitary failure will present all the classic features of myxedema. This type of patient may be differentiated from one with primary thyroid failure by investigation for failure of the other endocrine glands, roentgenologic examination of the sella turcica for tumor and examination of the visual fields. It has been advocated that all patients with myxedema should have a roentgenologic examination of the sella turcica. In our opinion this is not necessary if the patient has had a previous thyroidectomy for Graves' disease or struma lymphomatosa, but should be done in those patients with associated amenorrhea or loss of libido and potency.

The patient with a nephrotic syndrome due to any cause may offer initial difficulty in differential diagnosis especially when the basal metabolic rate and plasma cholesterol are determined. However, a careful history, presence of anemia, albuminuria and reversal of the albumin-globulin ratio should easily differentiate the disease from thyroid failure.

TREATMENT

After the diagnosis has been well substantiated the nature of the disease and the necessity of taking thyroid extract permanently should be explained to

the patient. Despite the many new types of thyroid preparations we still find uncoated U.S.P. thyroid extract to be the best standardized preparation so far. Enteric coated or enseal coated thyroid tablets should not be used. We have repeatedly seen patients on liberal doses of coated thyroid tablets who still exhibit all the signs of myxedema.

In the absence of a history of cardiac failure or angina pectoris a daily dose of 1 gr. of U.S.P. thyroid extract is prescribed for one month. If at the end of one month the patient has noted no signs or symptoms of cardiac failure or angina pectoris, the thyroid medication is permanently increased to 2 gr. per day. If angina pectoris occurs, the dose of thyroid is reduced to ¼ or ½ gr. per day. Such a small dose of thyroid will usually cause most of the signs and symptoms of myxedema to disappear although the patient may continue to have evidence of thyroid failure.

The symptoms and signs will usually be resolved within a month. To substantiate the diagnosis further, the basal metabolic rate and blood cholesterol should again be determined after two months. In addition, it is our practice to give the patient after the two month return visit, a letter certifying that a diagnosis of thyroid failure has been made on the basis of clinical signs and symptoms, laboratory tests, and response to treatment. We reemphasize the importance of permanently taking thyroid extract. The certifying letter is given so that the patient will not have the thyroid extract stopped in the future by another physician, as we have done, to prove or disprove a diagnosis of thyroid failure.

SUMMARY

Thyroid failure may be due to extrinsic causes or intrinsic disease. In some instances, extrinsic cause and intrinsic disease combine to produce thyroid failure. Hypothyroidism is likely to occur postoperatively in those patients with Graves' disease who have an abnormal degree of lymphocytic infiltration in their partially resected hyperplastic thyroid glands and also in those patients who have had a partial resection of a Hashimoto's goiter. Both of these groups of patients should be given thyroid extract postoperatively and the administration should continue for the rest of their lives. The cause of most spontaneous myxedema is probably some form of lymphocytic thyroiditis rather than simple atrophy.

The diagnosis of thyroid failure will probably be missed unless the clinician can keep in mind the characteristic features of the "myxedema facies." This type of facies is characterized by a pale yellowish-tinged color, puffiness of the entire face, especially over the upper eyelids and portion of the cheek above the angle of the jaw, and also the presence of an apathetic or listless expression with thickened lips. The delayed biceps or ankle jerk reflexes are specific for myxedema and when present are a valuable aid to diagnosis.

In our experience the determinations of the basal metabolic rate and plasma cholesterol before and after treatment with thyroid extract are still the best and most practical aids for the diagnosis of thyroid failure.

Such conditions as low metabolism without hypothyroidism, anterior pituitary failure, and nephrosis must be carefully differentiated from thyroid failure.

Treatment of thyroid failure should be administered in the form of uncoated U.S.P. thyroid extract for the rest of the patient's life.

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THE PEDICLED SKIN FLAP

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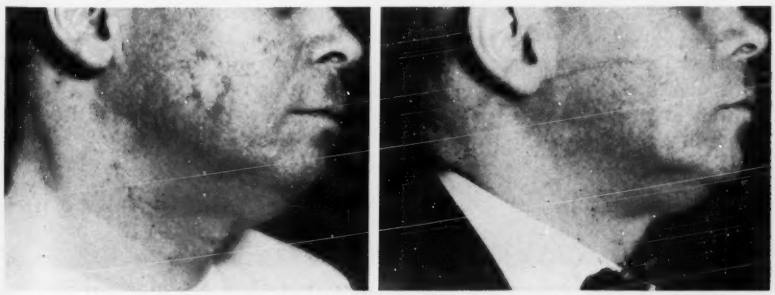
THE pedicled flap, commonly used by the plastic surgeon in the reconstruction of skin and soft tissue defects, differs from the so-called free skin graft in two ways: first, it consists of all of the layers of the skin, with a significant amount of attached fat and subcutaneous tissue; and second, it carries its own blood supply from one or more segments of its periphery.

This type of flap has been used since the earliest recorded days of medicine. As early as 800 B. C. Sushruta, the father of Hindu surgery, described in detail operations for the reconstruction of lost noses and lips by means of flaps from the cheek. Tagliacozzi in 1597 presented a method of reconstructing the nose by means of a pedicled flap from the upper arm; a technic known today as the Italian method and still employed. The use of a forehead flap for nasal reconstruction was developed on the basis of reports of such operations brought to England from India early in the 19th century. Since then, the utilization of such pedicles in reconstructive surgery has become increasingly common, particularly following Gillies' development of the tubed flap within the last 30 years.[1]

In general, it can be said that the pedicled flap should be used in preference to some other type of replacement material wherever skin coverage plus additional soft tissue with or without new blood supply are required. For example, defects over bony prominences such as the elbows and tuberosities of the ischium, where the possibility of damage to the covering structure is significant, require soft tissue padding in addition to the superficial skin. This is particularly true of the sole of the foot which is exposed to constant severe trauma in the course of normal activity.

Avascular tissues such as bone and cartilage are usually unable to support a free skin graft. Their coverage is best obtained by means of pedicles carrying



(a) (b)

Fig. 1. Typical example of repair by local flap of defect produced by removal of basal cell carcinoma; flap undermined widely and advanced to new position. (a) Preoperative. (b) Postoperative.

their own blood supply. The repair of severe, chronic, radiation lesions in which deep scarring makes total excision impossible, presents the same problem because of severe loss of vascularity.

Particularly during a war, compound injuries occur in which surface defects are associated in continuity with bone, nerve or tendon injuries for which further surgery is necessary. Since the success of any healing process depends upon an adequate blood supply, it is imperative that superficial scar be replaced with pedicled skin and soft tissue before surgical procedures are carried out on the deep structures. The healing of fractures and the vascularization of bone and cartilage grafts demand similar blood-bearing covering material. Finally, the demands of cosmetic restoration may require the presence of soft tissue and new blood supply as well as skin. This becomes obvious when one considers the problems inherent in building a nose, the contour of which depends not only on bony and cartilaginous support, but also upon the rounded contours of the tissue itself.

Considerable thought must go into the preparation of a pedicled flap prior to its manipulation. Careful planning of the flap with particular reference to the blood supply to be carried from its base is essential, with the further consideration that the transfer of this pedicle must not create a second prominent deformity in the course of repairing the original one. A flap may be of any size providing that its length to width ratio is calculated on the basis of available blood supply. In most instances one hesitates to construct a pedicled flap vascularized by a single source, in which the length of the flap is more than three times its width. Deficiencies in potential circulation may be corrected by a procedure known as "delaying," which consists of progressive elimination of blood supply, except at the proposed base of the flap, by means of surgical incisions around most of its periphery. Since maximum compensation in the remaining vessels takes place within about three weeks, a reasonably accurate estimate of the optimum time for utilizing the flap can be made. Any number of individual "delays" can be carried out to assure the viability of the flap at the time of transfer. It is most important that the flap be handled delicately during these procedures to avoid damage to barely viable tissue. Hemostats are used on the flap only to control vigorous bleeding. The pedicle is manipulated with fine hooks in its subcutaneous layer to avoid compromising the circulation more than absolutely necessary. The flap when transferred to a recipient area is sutured in place in such a way as to avoid any tension across the suture line or along the course of the pedicle itself. To assure this absence of tension, flaps are usually constructed somewhat larger than apparently necessary to allow for inevitable shrinkage.

TYPES OF FLAPS

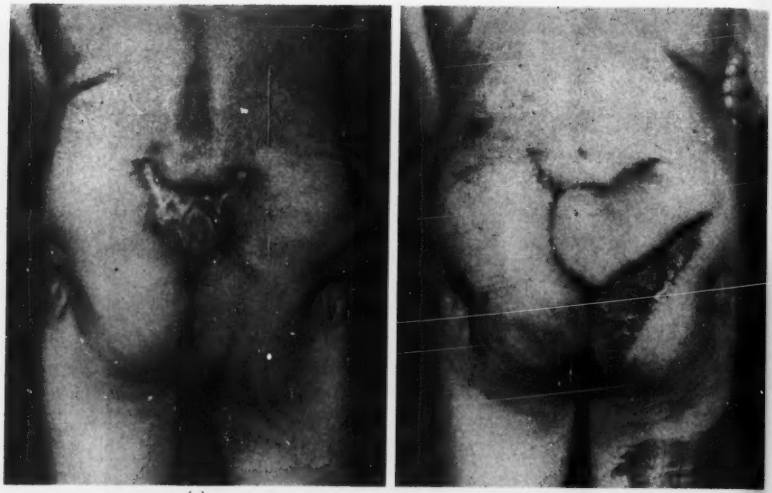
Many types of flaps have been described. In general they fall into two categories: *adjacent* and *distant*.

The *adjacent flap* is prepared in the immediate neighborhood of the area to be covered, and transferred to its new location in such a way that the remain-

(b)

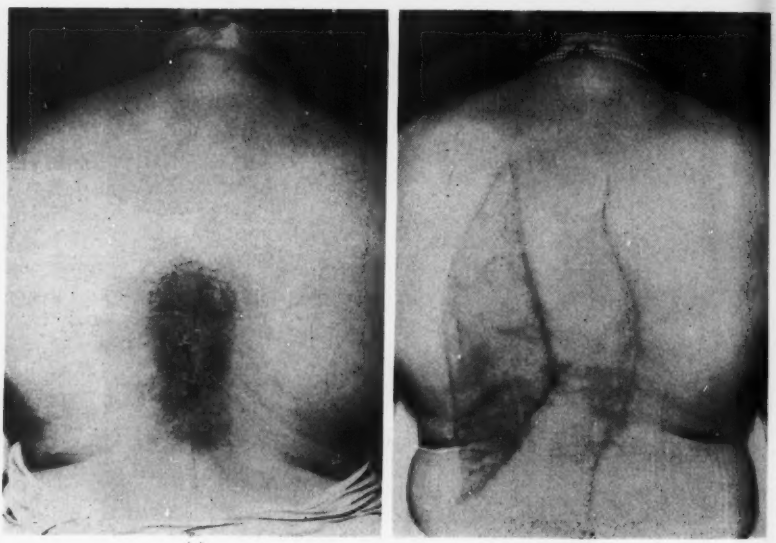
(a)

Fig. 2. (a) and (b) are two examples of repair by pedicled flap of full-thickness defects of scalp in which the flap donor site has been covered with split skin.



(a) (b)

Fig. 3. Sacral decubitus ulcer containing squamous carcinoma treated by excision, and transfer of blood-bearing pedicled flap. (a) Preoperative. (b) Postoperative.



(a) (b)

Fig. 4. (a) Severe radiation lesion of back following treatment of vertebral hemangioma. (b) Double-pedicled flap transferred to recipient site; donor defect covered with split skin graft.

ing donor defect produces little cosmetic or functional deformity (fig. 1). If the donor defect cannot be closed primarily, a small skin graft is usually satisfactory (fig. 2 and 3). The terms "advancement," "sliding," and "rotation" as applied to this type of flap merely imply the direction and means of transfer to the recipient site. These flaps may be based on a single or double blood-bearing pedicle, the latter providing additional insurance against inadequate circulation (fig. 4).

The *distant flap* is one in which the donor site is not in the immediate vicinity of the defect to be repaired. Thus, one or more transfers are necessary,

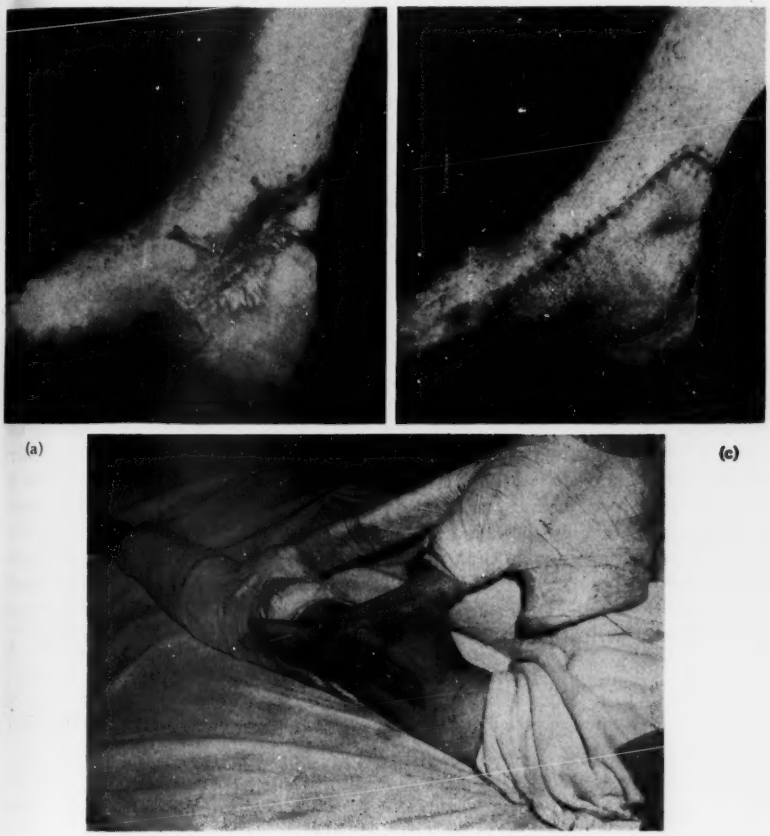


Fig. 5. (a) Scar deformity of ankle following compound fracture with skin and soft tissue avulsion. (b) Flap from opposite thigh in process of being transferred to defect. Two "delays" had been carried out prior to this stage. (c) Final result, showing good coverage which will permit additional bone surgery if necessary.

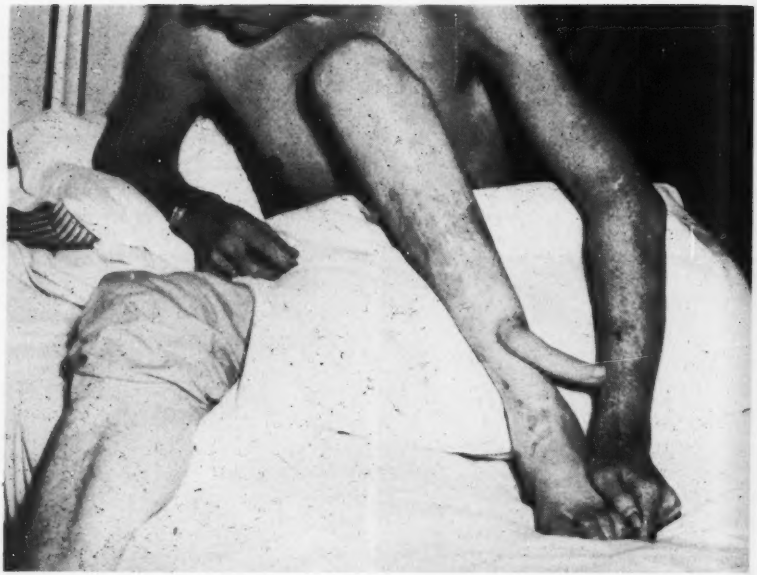


Fig. 6. Tubed pedicle carried on the wrist from abdomen to leg defect.

during each of which new blood supply is acquired, to reach the recipient area. One such transfer may be all that is necessary, as for example, in the use of the thigh flap to cover a defect of the leg or foot (fig. 5). If additional transfers are required, a mobile part of the body, usually the wrist, is used as a carrier to simplify the procedure. In this instance, the flap is often tubed to eliminate drainage and infection always associated with any raw surface (fig. 6). The only disadvantage to the tubing of a flap is the increase in total time required to complete the procedure. Occasionally, when a distant transfer is to be made over a short distance, the flap may be moved caterpillar-fashion. This procedure is impractical when more than two or three transfers are required.

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ENDOMETRIOSIS OF THE BLADDER

A Report of Three Cases

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A REVIEW of the literature reveals 68 cases of endometriosis of the urinary bladder. It is difficult to estimate the incidence of this lesion, since in the past cystoscopic inspection of the bladder has not been routinely employed in the presence of pelvic endometriosis.

Masson[1] in a review of the records of 576 patients in whom diagnoses of endometriosis had been established cited two instances in which vesical lesions were demonstrable; while Seitz[2] reported three patients with bladder involvement in 65 cases of pelvic endometriosis.

In a series of 530 consecutive cases of pelvic endometriosis observed at the Clinic, vesical lesions were present in three; a fourth instance, not as yet confirmed by biopsy, is not included. In reports reviewed in the literature, it is not stated whether cystoscopy was a routine procedure or employed only when symptoms deemed it advisable. In this series of 530 cases, cystoscopic examinations were performed only in patients with symptoms referable to the bladder. It is our purpose to report these three cases of vesical endometriosis and comment briefly on current therapeutic trends.

CASE REPORTS

Case 1. A woman, 43 years of age, Para I, gravida I, entered the Clinic in June 1952 with complaints of urgency, frequency, dysuria, and occasional severe pain following micturition. The symptoms had been observed for 15 years, beginning on the second or third day of each menstrual period and abating in approximately one week. Terminal hematuria had been noted on one occasion in May 1952, a month before admission. There were no other symptoms referable to the pelvis.

The past history was noncontributory except for her inability to become pregnant following the birth of one child.

General examination revealed normal findings.

A mid-cycle pelvic examination was normal except for a somewhat cystic left ovary. The intravenous urogram showed excellent function of both kidneys, while routine laboratory studies were normal.

On cystoscopic examination, clustered vesicles of various sizes were observed on the posterior wall of the bladder. The vesicles ranged in color from red through blue to a black tinge. A cup biopsy was performed and microscopic study of the tissue revealed endometriosis (fig. 1).

Following these reports, treatment was immediately instituted consisting of external roentgen therapy, 1000 r of 200 K.V. radiation to the anterior and posterior pelvic

fields, calculated to produce castration. With the cessation of the menses, the patient experienced complete relief of symptoms.

Cystoscopic examination approximately one month after treatment revealed only one extremely small vesicle located in the site previously occupied by the extensive lesion.

Case 2. A woman, 42 years of age, entered the Clinic in January 1951, complaining of pain in the region of the right kidney. The pain which previously consisted of attacks of renal colic had changed to a constant, steady, dull-aching character during the eight weeks preceding admission. She also experienced frequency, dysuria, and urgency. There was no history of hematuria, and she had noted no relationship between her symptoms and the menses. A pelvic operation had been performed elsewhere in 1930, the nature of which could not be determined. In 1950 an exploration of the right kidney had been undertaken elsewhere, the nature of which could not be ascertained; but the symptoms had persisted and menses ceased shortly after this surgical procedure. There was no history of pregnancy.

Physical examination revealed diffuse enlargement of the thyroid gland. The right kidney was palpable. On pelvic examination a firm fixed mass, 3 by 5 cm., was palpated in the right adnexal region. This was believed to represent the right ovary.

Intravenous urographic study demonstrated only impairment of function of the right kidney.

The routine laboratory studies were normal. *Escherichia coli* was isolated from the urine secured by catheterization.

The cystoscopic examination disclosed a flat irregular tumor, the size of a dollar, just above and encroaching on the right ureteral orifice. A No. 4 ureteral catheter encountered an obstruction 2 cm. from the orifice. Following cystoscopic survey, a specimen was secured for biopsy by means of the resectoscope, and a diagnosis of endometriosis established by microscopic study.

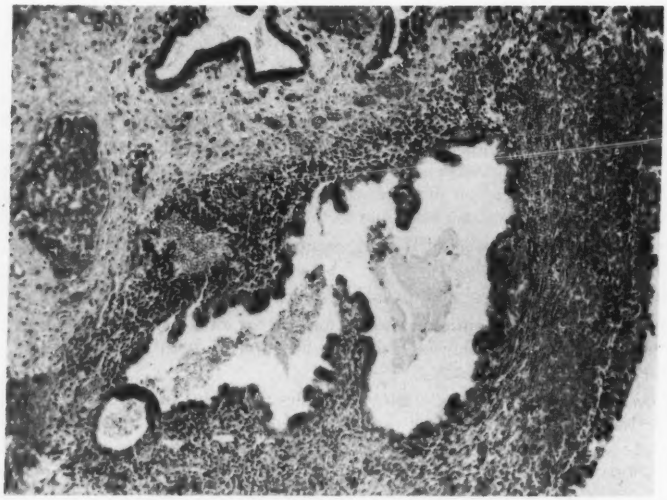

Fig. 1. Microscopic section showing area of endometriosis of bladder.

The treatment instituted consisted of external irradiation, 1000 r of 200 K.V. to the anterior and posterior pelvic fields; however, the pain in the right renal area persisted. Three months following the administration of roentgen therapy the right ureter was dilated and cystoscopic examination revealed a pronounced regression of the bladder lesion. Six months later a ureteral calculus, not demonstrable at the time of the initial examination, was removed, and the ureter again dilated. There was no residuum of the intravesical endometrioma discernible and the patient has remained free of symptoms for more than two years.

Case 3. The patient, a woman 31 years of age, Para 0, gravida 0, entered the Clinic in October 1946, complaining of suprapubic pain, urgency, dysuria, and frequency. The symptoms had been present for 20 months, beginning premenstrually and terminating with the onset of flow. She had never detected the presence of blood in the urine. Six months prior to admission a left salpingo-oophorectomy had been performed for endometriosis. Incomplete treatment for asymptomatic syphilis had been instituted in 1940.

The physical examination revealed a fixed, dilated, right pupil and moderate enlargement of the heart with a grade II aortic systolic murmur. The pelvic examination was normal.

The blood and spinal fluid Wassermann tests were positive. Other laboratory findings were normal.

Intravenous urographic studies demonstrated the function of the kidneys to be satisfactory. *Proteus bacillus* was isolated from the catheterized bladder urine.

Cystoscopic examination revealed a single bluish-colored vesicle, the size of a cherry, in the region of the left ureteral orifice. A specimen for biopsy was secured and on microscopic study demonstrated glandular structures with old hemorrhage. The lesion was observed to increase in size with each menstrual flow.

Treatment was begun with roentgen therapy, 1000 r, 200 K.V. to the anterior and posterior pelvic fields. The menses ceased but her symptoms persisted.

In July of 1949, approximately three years after entry, surgical intervention was instituted and an endometrioma of the bladder was resected. A remaining cystic portion of the left ovary was removed.

Following this procedure the symptoms were completely alleviated. She was again observed in 1953, four years later, at which time a hernia was repaired. There were no clinical symptoms referable to the bladder, and cystoscopic examination revealed normal findings.

DISCUSSION

In order to establish the diagnosis of endometriosis it is essential to investigate thoroughly patients with urinary tract symptoms. The symptoms of bladder involvement by endometriosis are variable and hematuria may not be present. Moore[3] has stated it is evident in less than one third of the cases. In the three cases reported here, blood was detected in the urine in one instance; also it is obvious that hematuria may be masked by the menstrual flow. Any symptom of vesical irritation should be viewed with suspicion if it is concomitant with menstruation. Occasionally, symptoms subsequent to ureteral obstruction will be present (case 2) or pelvic pain may be the only complaint.

Generally, diagnoses will be facilitated by cystoscopic examination during the menstrual flow, viewing the bluish-colored vesicles on the bladder wall. In some instances the characteristic vesicles are not evident and the lesion resembles a primary tumor of the bladder (case 2).

Ockuly and Helwig[4] believe that a diagnosis can be made in only a small per cent of the cases by biopsy. The cup biopsy is preferable to one secured by the resectoscope with coincident tissue destruction by the cutting current.

It is known that in general pelvic endometriosis, the symptoms frequently have no relation to the extent of the disease. Undoubtedly, this is also true in the bladder, but due to constant change in vesical dynamics, lesions in this location are often more symptomatic. Bladder lesions may coexist with generalized endometriosis involving other pelvic organs, or may occur without other evidence of the disease.

Numerous articles have appeared in the literature concerning the origin and nature of endometriosis,[5-7] but in this paper we will not consider this aspect of the disease.

The approach to therapy for endometriosis of the bladder is not unlike that instituted for the disease when located elsewhere. Treatment must be individualized and planned to meet the patient's needs and interests.

In the past several years there has been an increasing trend towards conservatism in treating endometriosis, irrespective of its location. When selecting the therapeutic program not only should the physical status of the patient and the extent of the lesion be considered, but equally important are the age, marital status, parity, and the desire for pregnancy.

Isolated bladder lesions may often be treated with local fulguration, since malignant degeneration, although reported, is unusual and complete destruction may be accomplished. When the lesions are more extensive they may be amenable to local resection. In instances in which the lesion is in close proximity to the ureteral orifice or situated on the trigone, irradiation of the ovaries may be the most conservative approach in a woman who is near the end of menstrual life. However, if the same lesion occurred in a younger person, local resection with reimplantation of the ureter into the bladder might render it possible for the patient to achieve a much desired pregnancy and also avoid the sequella of castration.

When the diagnosis of endometriosis is made and other pelvic findings suggesting widespread disease are noted, it may be assumed that all are associated with endometriosis. However, this does not preclude the possibility of other coexisting pelvic disease. It would seem that the wisest course in a patient whose general condition is satisfactory consists of operative evaluation with resection of the pelvic or bladder lesions if possible. Removal of the ovaries will of course be followed by regression of the lesions. By open operation it is usually possible to perform the surgical procedure most compatible with the patient's needs and still combat the disease. An endometrioma of the bladder may be resected or localized lesions in the pelvis removed, permitting the menses to continue and pregnancy to be achieved.

In other instances, if surgery fails or if it is impossible to remove the ovaries and uterus, we may then employ irradiation secure in our knowledge of the entire situation. In a patient, who is a poor surgical risk, primary irradiation may represent the most conservative approach.

The newer medical treatments consisting of estrogen therapy, as proposed by Karnaky,[8] and testosterone, cited by Schmitz and Towne[9] and Siegler,[10] may be of some benefit in selected cases.

Karnaky reported favorable results after the treatment of 37 women with large doses of stilbestrol. The rationale of this therapy is thought to be a suppression of the anterior pituitary gland with subsequent inhibition of ovarian function. Stilbestrol, however, carries with it the undesirable effects of nausea and complications of bleeding. Although it is effective in the relief of pain the obtainment of any remarkable regression of the disease is doubtful.

Testosterone is capable of suppressing ovarian activity and may be effective in controlling the bladder lesions; but it is mainly useful as a stopgap measure and utilized to postpone temporarily the need for more radical therapy. It is stated that this treatment is disadvantageous because in many cases, prolonged suppression of symptoms can not be maintained without masculinization.

In the three cases of endometriosis of the bladder reported here, roentgen therapy was advised on the basis of the following considerations:

Case 1. A nurse, 43 years of age, requested castration by irradiation of the ovaries. She was acquainted with the sequella of castration, but was opposed to surgical intervention. After consideration of the patient's personality and desires, roentgen therapy was employed.

Case 2. The patient was an emotionally unstable woman, 42 years of age, who had not menstruated during the year prior to admission. The endometrioma of the bladder encroached on the right ureteral orifice producing sufficient obstruction to impair the drainage of urine from the right kidney. As the menstrual life was already terminated, roentgen therapy appeared to be the treatment of choice.

Case 3. This woman, 31 years of age, had a diagnosis of syphilis made six years prior to our examination. The treatment was inadequate as evidenced by the blood and spinal fluid Wassermann tests being positive. Six months previously a left salpingo-oophorectomy had been performed elsewhere for endometriosis. Cardiac enlargement was present with a grade II aortic systolic murmur.

In view of these findings roentgen therapy was employed. In contrast to the results obtained in cases 1 and 2, pain and discomfort persisted. Three years later surgical intervention was required which afforded complete relief of symptoms.

It is interesting to note this patient was managed before the recent surge towards conservatism in the treatment of endometriosis.

SUMMARY

1. Three cases of endometriosis involving the urinary bladder have been discussed and conservatism in the management of these patients emphasized.

2. Local fulguration or open operation for endometriosis of the bladder are the treatments of choice.

3. Irradiation should be reserved for the patient who is a poor surgical risk, or upon failure of the previously mentioned treatment.

4. The role of estrogenic and androgenic therapy in these cases is as yet to be determined.

Acknowledgment

We wish to express our thanks to Dr. James S. Krieger of the Department of Gynecology for his comments.

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REOPERATED CONGENITAL MEGACOLON

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THE study of the pathology of congenital megacolon by Whitehouse and Kernohan[1] and Swenson's physiologic and surgical studies[2-4] form a logical approach to adequate therapy of the disorder best known as congenital megacolon or Hirschsprung's disease.[5] We viewed with some misgivings the recent report of State[6] who advocated retention of the major portion of the rectum as part of the surgical correction of this disorder.

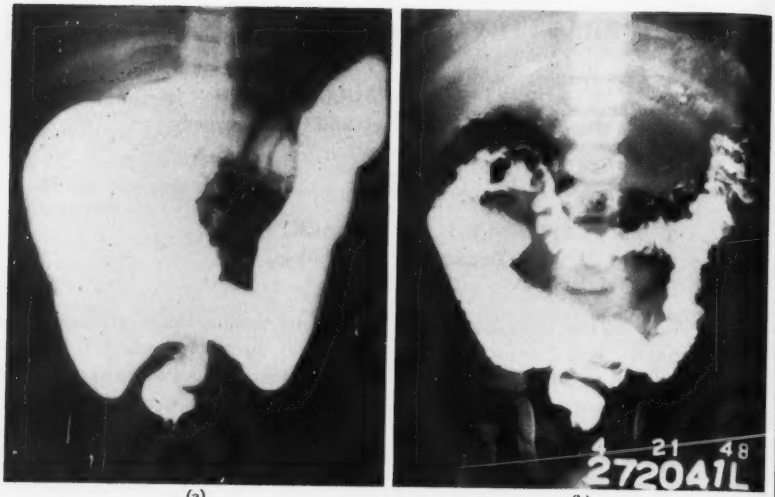
We have recently encountered a case in which the distal sigmoid and rectum containing an aganglionic segment had been retained, and reoperation was required.

CASE REPORT

Clinical Features

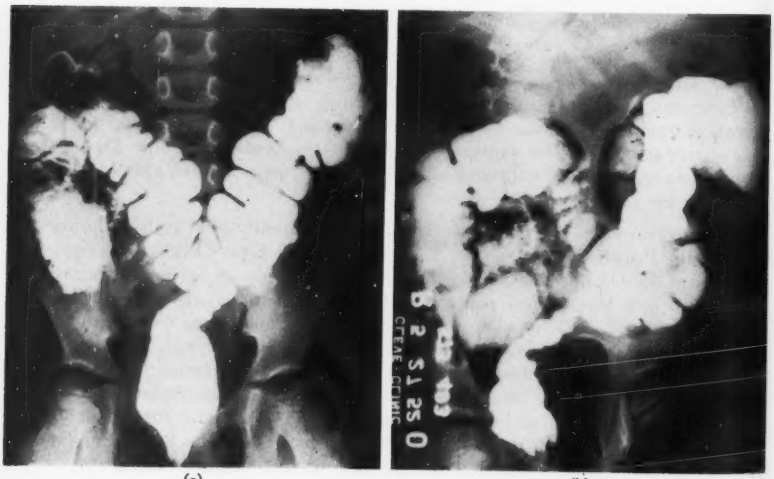
The patient, a two year old white boy, was first seen at the Clinic on April 29, 1949. His birth had been uneventful, but on the fourth day following delivery abdominal distention was noted which was relieved by enemas. The child then had six to eight loose watery stools a day for the first six months of life. At about the end of the first year the diarrhea was replaced with obstinate constipation, requiring the aid of high colonic irrigations for evacuation.

Physical examination revealed the boy to be in apparently good health. The abdomen was slightly distended and a putty-like mass filling the right flank was palpable. The rectum was dilated and contained a large fecal impaction. A barium enema revealed a narrowed area in the distal sigmoid which showed little change in the filled colon and evacuation films. The colon proximal to the narrowed area was greatly dilated, particularly in the cecum and ascending colon (fig. 1). A diagnosis of Hirschsprung's disease was made, and on May 13, 1949, 14 days after initial examination, a laparotomy was performed by the late Dr. Thomas E. Jones. The lower descending colon and sigmoid appeared greatly dilated with a transition zone to normal size about 4 inches above the peritoneal reflection. The dilated bowel was resected by the Rankin modification of Mikulicz's technic. The postoperative course was uneventful and on the fifth day a spur clamp was applied. Three months later, the colostomy was closed, and two weeks following this the diarrhea reappeared; the patient had three to six watery stools daily. Five months postoperatively the child became mildly constipated with a spontaneous bowel movement every third day. At this time no fecal impactions were noted and the remainder of the physical examination was normal.



(a) (b)

Fig. 1. Barium enemas in 1949. (a) Filled. (b) Evacuated. A constantly narrowed area is present in the distal sigmoid; the colon proximal to this area is greatly dilated, particularly in the cecum and ascending colon.



(a) (b)

Fig. 2. Barium enemas in 1952. (a) Filled. (b) Evacuated. The rectum is slightly narrowed at the rectosigmoid. Proximal to this area there is a segment of constant narrowing. Proximal to this narrowed area the colon is again dilated. Postevacuation film shows retention of nearly all the injected barium.

Three years following resection, the child returned because of sudden cessation of bowel movements with no natural evacuation for one month. Abdominal distention and cramps had been present during this time and enemas were given daily. His appetite had decreased and there had been some weight loss. The child was readmitted to the hospital, and in April 1952 proctosigmoidoscopic examination to 13 inches revealed a normal rectum and lower sigmoid colon with a widely patent anastomosis in the region of the old colostomy closure. The general physical examination was otherwise normal. No fecal masses could be palpated. A barium enema revealed the rectum to be slightly narrowed at the rectosigmoid, and for approximately 5 cm. proximal to this area there was constant narrowing of the bowel. The colon proximal to this narrowed area was considerably dilated. Postevacuation films showed retention of nearly all the injected barium (fig. 2).

At surgery the closed colostomy was found intraperitoneally located about 5 inches above the pelvic floor. There was minimal dilatation of the colon above this point and the terminal sigmoid below appeared to be normal. A full thickness biopsy of the wall of the colon proximal to the anastomosis, examined by frozen section technic,[7] revealed the presence of ganglia. The pedicle containing the inferior mesenteric artery and vein was then ligated near the lower border of the third portion of the duodenum, and the rectum was freed from its attachments in the pelvis, keeping the dissection as close to the bowel wall as possible. The resection and pull-through technic with anastomosis was carried out as described by Swenson. The anastomosis was made with single layer of 000 chromic catgut sutures. Watery bowel movements were noted on the third day and at least one bowel movement a day was noted thereafter. On the eighth postoperative day a small pre-sacral abscess drained spontaneously through the anastomosis; the child was discharged on the 21st day.

An attack of high intestinal obstruction, necessitating laparotomy with lysis of an adhesive band occurred in September 1952, five months after operation. Otherwise, the child's progress was satisfactory. A normal bowel habit was established and enemas and cathartics have not been necessary.

Pathologic Features

The original surgical specimen (May 1949) consisted of a flask-shaped segment of large intestine without attached mesentery, 20.0 cm. in length (fig. 3). The serosal surface was smooth, glistening, transparent, and appeared slightly edematous. The opened specimen was dilated to a circumference of 7.0 cm. at the proximal line of resection, gradually increasing to a maximum diameter of 12.0 cm. near the distal line of resection; finally, there was moderately rapid narrowing of the bowel to a circumference of 5.0 cm. at the distal line of resection. The bowel wall was thickened (up to 4 to 5 mm.) especially in the distal half. The mucosa was yellowish-tan in color and revealed no ulceration; in the proximal end of the bowel the mucosal folds were normal, but were thickened in the distal half.

Microscopically, multiple sections through the thickened and dilated portion of the bowel revealed a muscularis externa that was several times its normal thickness but with layers arranged in the usual fashion. The tissues were somewhat loosely arranged, suggesting edema. Myenteric ganglia were present in both the submucosa and between the circular and longitudinal muscle layers. They appeared to be slightly decreased in these areas, but this was relative and only apparent because of the large increase in size of the muscular layers.

The pathologic diagnosis was megacolon, probably of congenital type.

The operative specimen at the second operation (April 1952) consisted of 16 cm. of colon, including approximately 6.0 cm. of rectum and an old, well-healed anastomotic line. The specimen measured 7.5 cm. in circumference. The internal surface revealed a normal appearing mucous membrane. This entire specimen was serially blocked at approximately 0.7 cm. intervals. Sections of the colon adjoining the proximal line of old anastomosis revealed normally distributed ganglia.

The distal 7.0 cm. of the specimen (chiefly rectum) was the most important histologically. It must be divided into a proximal and distal segment. The proximal segment, 4.0 cm. in length, revealed moderate thickening of the muscularis externa, numerous bundles of nerve fibers between muscular layers and in the submucosa, but in no section from this area could myenteric ganglia be identified. It is of note that the muscularis mucosae in the aganglionic area was uniformly hypertrophied to a thickness approximately four times normal (fig. 4, a and b). The distal segment, 3.0 cm. in length, showed thinning of the muscularis mucosae to normal and a reappearance of myenteric ganglia. This change occurred gradually in the most proximal 1 cm. of this portion. The most distal 2.0 cm. portion of this segment then possessed a normal muscularis mucosae and a normal distribution of myenteric ganglia (fig. 4, c and d).

The pathologic diagnosis was congenital megacolon, previously operated.

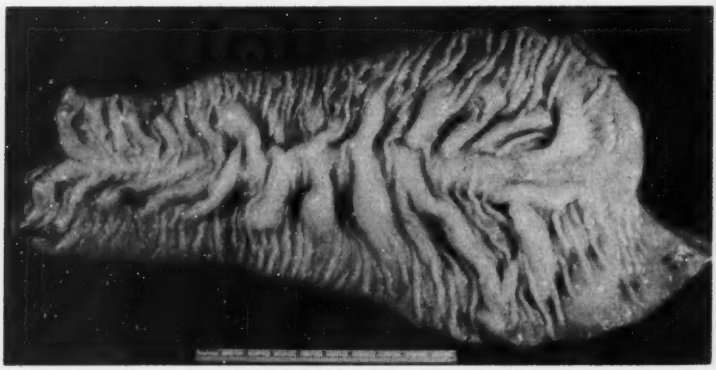


Fig. 3. Specimen of original surgical resection. Note flask shape and hypertrophied mucosal folds.

COMMENT

Congenital megacolon may be defined as the failure of normal bowel and rectal function dating from birth, associated with extreme dilatation of all or part of the colon with retention of feces and gas, and absence of the ganglionic elements over varying areas of the rectum and sigmoid colon. Hirschsprung's classic paper in 1887 aroused considerable interest in this condition but etiology and pathologic physiology remained obscure until recently. In 1948, Whitehouse and Kernohan[1] published a detailed histopathologic study of the rectum

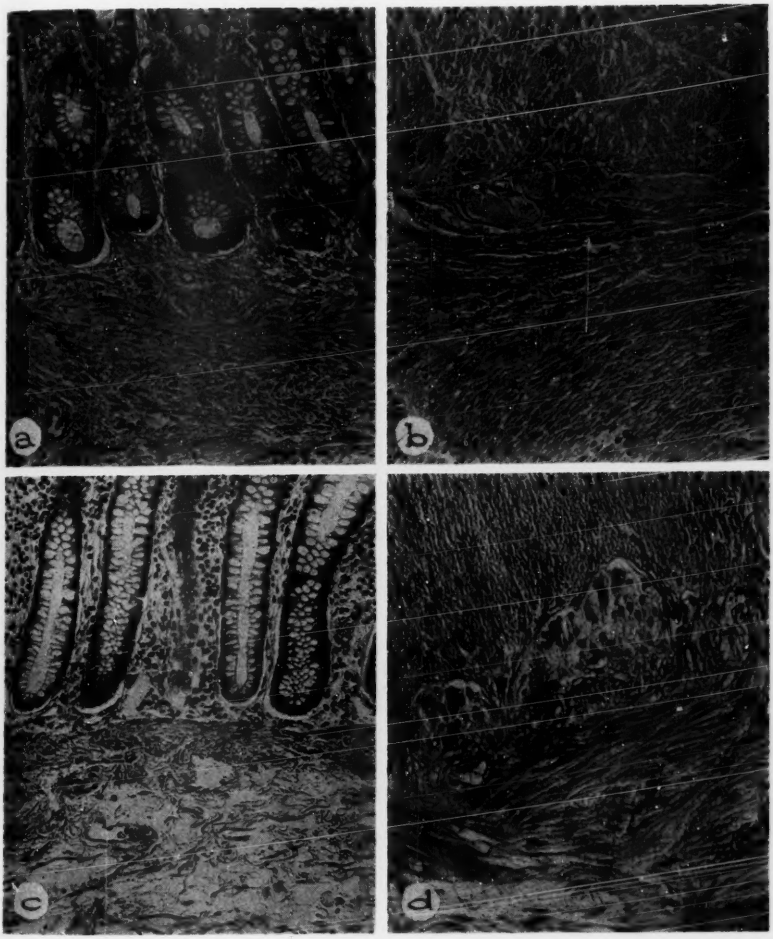


Fig. 4. Histology of portion of rectum removed at last resection. (a) and (b) Aganglionic area. (c) and (d) Ganglia-containing area. (a) Marked hypertrophy of the muscularis mucosae in aganglionic segment. X 130. (b) Representative area between layers of muscularis externa. Note absence of ganglia with presence of large nerve fibers. X 180. (c) Normal muscularis mucosae in distal rectal segment where ganglia were present. X 130. (d) Normal myenteric ganglia in distal rectal segment. X 180. Hematoxylin-eosin-methylene blue stain.

and colon of 11 patients. Their findings were: (1) absence of the myenteric ganglia of the rectum with a transitional zone at the rectosigmoid or extending upward wherein altered numbers of normal ganglia could be found; (2) normal ganglia in the remaining colon; and (3) the presence of closely packed non-myelinated nerve trunks between the longitudinal and circular muscle layers in the aganglionic segment of the bowel. Our case adheres closely to these morphologic features. However, in none of their cases was mention made of the reappearance of ganglia in the most distal portion of the rectal segment. They also made no mention of the marked hypertrophy of the muscularis mucosae in the aganglionic segment, which finding has been a rather constant feature of our surgically treated cases of congenital megacolon.

Many surgical procedures have been described for the treatment of congenital megacolon. None of them were truly satisfactory until Swenson and his co-workers reported their contributions in 1948. Swenson demonstrated disordered peristalsis in the rectosigmoid or rectum at the lower limit of the area of colonic dilatation and believed these parts to be physiologically obstructing. We believe that the hypertrophy of the muscularis mucosae at this level may be interpreted as further evidence of altered muscular activity within this area. By a modification of Maunsell's[8] operation, Swenson was able to resect the aganglionic rectum and rectosigmoid with preservation of the sphincters. The success of the Swenson operation depends upon total removal of those portions of the rectum or lower colon wherein no myenteric ganglia can be found. We have screened biopsies of these colons by frozen section technic to determine presence of myenteric ganglia.

The less radical procedure recently advocated by State,[6] *ie.* resection of narrowed sigmoid and upper rectal segments in addition to the proximal dilated colon with anastomosis between normal appearing colon and upper rectum, may be doomed to eventual failure since the aganglionic area may not be completely removed.

SUMMARY

A case of congenital megacolon is reported in which reoperation was performed because of initial retention of the aganglionic upper rectal segment. It is emphasized that single resection of the dilated, hypertrophied colonic segment alone may temporarily restore the patient's bowel function to a normal state even for a period of several years. However, retention of the aganglionic segment can eventually result in physiologic obstruction and recurrent megacolon, necessitating further surgery.

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ANESTHESIA FOR MITRAL COMMISSUROTOMY

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THE margin of safety during anesthesia for mitral commissurotomy is extremely narrow. In most patients undergoing this operation, pathologic changes in the cardio-respiratory system have resulted in pulmonary hypertension. The major pulmonary arteries show atheromatous changes; the small vessels show various changes ranging from simple muscular hypertrophy to hyalinization and fibrosis. Unless these serious anatomicophysiologic changes are understood, anesthetic agents might cause death before valvulotomy is accomplished.

All of the ability and skill of the anesthesiologist is required to administer anesthesia successfully to one of these patients. Anoxia is prevented by supplying the alveoli with an oxygen-rich atmosphere; blood pressure is maintained and adequate respiratory excursions guaranteed. Most important of all the functions of the anesthesiologist are: determination and maintenance of the optimum plane of anesthesia for successful completion of mitral commissurotomy; and the use of the *least* amount of anesthetic agents and technic as possible. When deep levels are reached, the strain on the cardiac patient is too great for his compensatory ability; serious arrhythmias appear or a progressive bradycardia threatens cardiac arrest. Light anesthesia is characterized by its lack of influence upon the cardiac mechanism.

Adequate oxygenation must be guaranteed in all anesthetized patients. During intracardiac procedures, an already deficient oxygen saturation caused by anatomic changes of the disease itself is partially remedied by filling the alveoli with an oxygen-rich mixture. Full expansion of the lungs not only insures delivery of these mixtures but also accomplishes the removal of any carbon dioxide which may have accumulated. Distention of the lung will prevent collapse with the resultant loss of oxygenating capacity through the atelectatic portion. During intracardiac procedures, the myocardium is repeatedly irritated by manipulation, suturing and cutting. This irritation to the pathologic heart causes irregularities of rhythm which are held in abeyance by delivering blood totally saturated with oxygen to the coronary circulation.

To satisfy myocardial oxygen demands during anesthesia, there are three basic requirements which are accentuated by the particular needs of intracardiac surgery. (1) Sufficient oxygen content is required in the breathing mixture. The 20 per cent oxygen content of atmospheric air may or may not be sufficient for the patient's basal needs. During anesthesia a mixture of greater oxygen concentration is delivered, but a point is reached when further increase is ineffective in increasing the percentage of oxyhemoglobin. Therefore, during anesthesia it is necessary to deliver an anesthetic mixture of gases containing 20 per cent, but preferably 40 or 50 per cent oxygen. The decreased

cardiac output caused by a stenosed valve can be partially compensated by completely saturating the hemoglobin in the pulmonary circulation. (2) Adequate amounts of hemoglobin must be present in the blood to satisfy the demand of the tissues for oxygen. (3) An adequate circulatory pressure must be provided to distribute oxygen to the vital centers. Blood pressure must be maintained within normal levels.

Hypercarbia is prevented by the use of the carbon dioxide absorption technic. The lung is fully expanded by sufficient volume and pressure, aerating as much of the lung as is possible, consistent with good surgical exposure. Obliteration of the field by the lung is prevented by the use of surgical retractors. Any unexpanded areas of lung may simulate an arteriovenous shunt, producing hypercarbia and suboxygenation of the arterial blood. Without consideration of the physiologic limitations of the cardiac patient, such quantities might seem trivial.

The ideal depth of anesthesia is the lightest level that permits successful surgery.[1] George W. Crile[2] stated: "The paramount object of anoci-association is to reduce the toxic action of the general anesthetic and the traumatic factor of the operative manipulations to a minimum."

Major surgery requires: (1) preoperative sedation; (2) hypnosis or loss of consciousness; (3) analgesia; and (4) relaxation. Any agent or group of agents in minimal doses which meets these requirements and produces no real physiologic aberrations is satisfactory. In our hands no one single agent is capable, and we therefore use a group of agents in minimal doses to produce light planes of anesthesia.[2]

TECHNIC

Premedication. Demerol or morphine is administered two hours before surgery in doses according to weight and age. Atropine is not used in valvular surgery because the resulting tachycardia allows insufficient filling of the heart. One cc. of 1/2000 solution of prostigmin methylsulfate is injected subcutaneously one hour preoperatively. Nembutal is given orally three to four hours before operation.

Induction. All patients are induced in their respective rooms. Forty to eighty mg. of pentothal is injected intravenously; if the patient asks the purpose of the injection he is told it is routine medication. In no case is the patient informed that he or she is going to the operating room.

When sufficiently sedated, the patient is wheeled to the operating room. It is seldom necessary to use more than 80 to 100 mg. of pentothal during the interim.

Preparation in Operating Room. An infusion of 500 cc. 5 per cent dextrose containing 2 cc. neosynephrine[4] is started intravenously by extremely slow drip sufficient to keep the needle open. Electrodes are placed and the

continuous electrocardiogram started. Noise and talking in the operating room are held to a minimum, and the patient repeatedly reassured.

Intubation. The larynx and pharynx are sprayed with 4 per cent cocaine solution under direct vision. When small doses of pentothal are given, direct visualization of the larynx is quite easy. Laryngospasm results only when larger amounts of pentothal are given. But when 25 to 50 mg. is administered, the laryngeal reflex is not excited and the parasympathomimetic qualities of the drug not displayed.

With adequate cocainization, a Murphy endotracheal tube equipped with a Sander's cuff is inserted into the trachea and a gauze bite block placed between the incisor teeth.

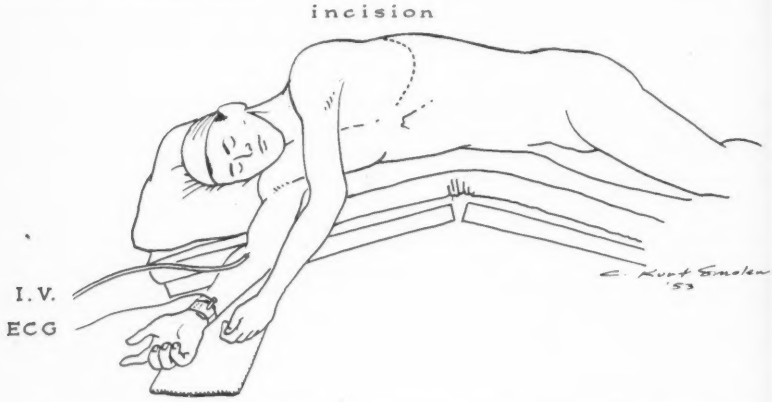

Fig. 1. Position of the patient for mitral commissurotomy showing sites of intravenous infusion and electrocardiograph electrode.

Positioning. Before other agents are given, the patient is placed in position by the surgical team. The right arm containing the intravenous needle is supported with an arm board extending from the table. The left arm, supported by a pad, is allowed to hang over the edge of the table and carries the scapula out of the surgical field.

Anesthesia. The level of hypnosis at this point varies but in the vast majority of cases, the patient will react to questioning by shaking his head and opening his eyes. Nitrous oxide (80 per cent) and oxygen (20 per cent) is started with a 5 L. flow. This overflow guarantees the percentage mixture, the nitrogen displacement with nitrous oxide, and the elimination of carbon dioxide. Variation of gaseous mixture is regulated according to the needs of the patient, but an oxygen concentration as high as possible is maintained at all times.

A mechanical respirator* can be interposed in the system. Curare is not necessary for its proper function and the patient will soon allow this machine

**The Rand Wolfe Respirator is used at the Clinic.*

to take over respiration. The rate is set at 15 per minute. This slow rate is entirely satisfactory and is more constant and reliable than manual compression. Using a Rochester Model Heidbrink, the escape valve is set at 15 to 18 mm. pressure. The respirator with overflow of anesthesia gases guarantees the patient sufficient respiratory excursions, safe gas pressures, adequate oxygenation, and frees the anesthesiologist's hands for other exceedingly important duties.

The level of consciousness is lowered by pentothal and nitrous oxide, but the patient is never reduced to anesthesia levels where response to questioning disappears. Complete cortical release is avoided and the thalamic and hypothalamic reflexes are obtunded by this degree of consciousness. The intercostal space is infiltrated with procaine 1 per cent. The patient is unaware of the skin incision and pleural and visceral pain is not perceived.

Manipulation of the lung occasionally incites coughing. Restraint must be exercised not to inject superfluous quantities of anesthetic agents into the delicate balance so far secured. If the surgical field is too active, 1 or 2 cc. of d-tubocurarine is injected intravenously. Within a few minutes diaphragmatic and mediastinal activity lessens or disappears. The patient is quiet and perceives no pain. All nocuous physical and psychic stimuli have been excluded from the brain. The surgical field is quiet and mediastinal flutter nonexistent under light pentothal-nitrous-curare anesthesia.

If the patient fails to respond to questioning, the level of anesthesia is too deep. The concentration of nitrous oxide in the nitrous oxide-oxygen mixture is lessened from the 80/20 to ratios as low as 50/50. Within a few minutes the anesthesia will lighten to an optimum plane. When the patient is questioned about pain or discomfort, he denies its presence with a shake of his head. For the occasional patient in whom analgesia is insufficient, pentothal may be the immediate choice. This agent not only renders the patient totally unconscious, but also initiates the parasympathomimetic activities of the drug. Demerol or morphine lowers the basal metabolic rate, sedates the patient and raises the tolerance to pain. These agents permit any intrathoracic manipulation.

Occasionally nembutal or seconal is substituted for pentothal in the induction of anesthesia. Parasympathomimetic action is not predominant in these agents. The effect upon the vagus is minimal and laryngospasm and bradycardia become less of a hazard. However, the slow onset of hypnosis and delayed maximal effect render the agents difficult to use. Minimal calculated doses may turn out to be insufficient or excessive. To prevent such errors, oral nembutal is given for premedication and two hours later pentothal induces the anesthesia by fractional dose administration. The effect of this ultra fast-acting drug in 40 to 80 mg. doses can be instantly determined. Seldom is this amount depressive, and it can be immediately supplemented if found insufficient. To create the exact state of hypnosis requires an agent which can be easily administered and the effects of which are immediately evident.

During the closing of the thoracotomy wound, it is frequently necessary to reinforce the general anesthesia by local wound infiltration with procaine.

The lungs are inflated by a continuous positive pressure varying between 5 and 20 mm. Hg. or prolonged positive pressure at the height of each inspiration, guaranteeing that no atelectatic areas remain. The thorax is then closed with this positive pressure until the "water-seal" suction apparatus is attached to a pleural catheter. The patient is moved from the right lateral position slowly and with great deliberation to avoid the hypotension which may be induced by position change. The respiratory tree is suctioned by inserting a catheter through the endotracheal tube. Long periods of suctioning are to be avoided. The patient at this time is fully awake and will cooperate by coughing. Extubation is performed as soon as is possible after the patient resumes the supine position. When the circulatory system is stable (characterized by adequate blood pressure and acceptable electrocardiographic readings) the patient is lifted to the bed and transported to the recovery room.

The tape recording electrocardiograph, a research model designed by Dr. W. L. Proudfit of the Department of Cardiovascular Disease at the Clinic, affords a continuous picture of the cardiac mechanism. The oscilloscopic screen depicts each contraction, and variation from normal can be instantly determined. The pulse is counted with each sweep of the screen. The continuous electrocardiograph of lead 2 on sound recording tape during surgery allows review by the internist at a later time.

DISCUSSION

Anoxia during anesthesia is to be avoided at all costs. Sufficient concentrations of oxygen are to be included in all anesthetic mixtures regardless of the type of surgery or the condition of the patient. Positive pressure respiration (manually or mechanically) sufficient to expand the lung during open thoracotomy and a circulation sufficient to carry an adequate supply of oxyhemoglobin to the tissues must be guaranteed. The mechanical respirator creates a simple respiratory cycle and can be adjusted as to rate and intrapulmonary pressure. By this means an adequate concentration of oxygen is delivered to the alveolar walls under sufficient pressure to fully expand the lungs rhythmically at a predetermined rate.

In the usual patient suffering from valvular stenosis, the red cell mass and hemoglobin are adequate. A tachycardia (which prevents adequate filling of the heart in diastole) and hypotension (stagnant anoxia) herald myocardial anoxia. Both are to be avoided with equal vigor to prevent changes in cardiac rhythm. Carbon dioxide retention is seldom a factor during this type of anesthesia. Soda lime absorption technics with a semi-closed positive pressure system minimize the probability of hypercarbia.

The level of anesthesia in many instances determines the outcome of the entire procedure. When the patient can react to questions and yet experiences no pain during commissurotomy, the ideal level is obtained. This statement sounds contradictory and inconsistent. During the actual commissurotomy, the patient shakes his head when asked if he is experiencing pain. Seldom is

discomfort experienced except when skin is sutured without procaine infiltration. Cardiac arrhythmias during this light anesthesia are seldom in evidence. Frequently pre-existing arrhythmias will revert to a normal rhythm during anesthesia.

Other anesthetic agents are not utilized because they are neither necessary nor indicated. Nitrous oxide is a relatively inert gas producing no systemic changes in the body. Its anesthetic effect is of short duration, being exhaled within a few minutes. Few if any other anesthetic agents can qualify in this manner. Nitrous oxide is nonexplosive and does not affect cardiac conductivity or rhythm. It permits extra-coagulation of bleeding points,[3] which decreases operating time tremendously by rapid control of skin bleeding. Conversely, ether will cause pulmonary hypersecretion and all the resultant pulmonary dysfunction.[5]

In many instances the patient undergoing commissurotomy has tolerated a decreased peripheral circulation for a number of years. Adequate circulatory pressure, however, must be maintained to satisfy myocardial circulation. After valvulotomy the peripheral circulation and cardiac output is increased. Hypotension ensues and may progress to alarming levels. Blood pressure readings under 80 mm. Hg. are not well tolerated as evidenced by electrocardiographic changes in rhythm and rate. If further exploration or manipulation of the heart is performed during this period, exaggerated electrocardiographic variations appear and may persist for alarming periods in spite of the usual therapeutic measures. A rapid infusion of the neosynephrine/dextrose solution (.2 mg. in 500 cc. dextrose) converts a dangerous hypotension to a safe level.

Bradycardia may be produced by anoxia, in which case re-evaluating the oxygen concentration of the mixture constitutes a cure. Atropine sulfate increases the rate to safer levels when it persists below 60 per minute.

Tachycardia seldom occurs when prostigmin is given preoperatively. In most cases, the pulse rate is between 60 and 70 and seldom reaches 100 before valvulotomy. After the stenosed valve is fractured, it is not uncommon for the pulse rate to increase to 90 to 100 per minute. Rates above this figure occur concomitantly with an uncontrolled hypotension.

Pronestyl is used preoperatively. During anesthesia 2 cc. of 2 per cent procaine hydrochloride (40 mg.) is administered intravenously to control an arrhythmia such as ventricular tachycardia. Repeated injections of 40 mg. may be needed before the irritability of the myocardium decreases. When the integrity of the myocardium is compromised, as evidenced by ECG tracings of ventricular tachycardia, the surgeon is requested to desist. The patient is allowed to rest. The lungs are fully expanded and the entire gaseous mixture renewed.

With this type of anesthesia serious complications have been absent. Transient paroxysms of arrhythmias occur during fracture of the valves only to disappear spontaneously when surgical manipulation ends. Frequently this hypersensitivity is controlled by prophylactic use of procaine hydrochloride intravenously immediately preceding valvulotomy. In the last 50 cases using

this type of anesthesia it has not been necessary to terminate any operation before a definitive procedure was accomplished. Cardiac arrest or other serious arrhythmias has not been encountered in this series.

Although the patients react to questioning during the valvulotomy, not one can recall anything about the operation. Usually they remember the venipuncture in their ward or room. Occasionally one remembers vaguely the bright lights of the operating room. None can recall the operation or pain.

SUMMARY

A system of pentothal-nitrous-oxide-curare anesthesia for mitral commissurotomy is presented; the salient feature of which is the light plane of anesthesia maintained with small doses of the agents. The patient is totally analgesic and amnesic, but will answer questions by shaking or nodding his head.

In no case in a series of 50 patients was it necessary to discontinue anesthesia before a definitive procedure was accomplished; nor was cardiac arrest encountered.

All patients were awake and extubated at the end of the operation.

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PECTUS EXCAVATUM: SURGICAL TREATMENT

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THE unsightly deformity of pectus excavatum, or "hollow chest," is an acquired lesion. It usually occurs in healthy infants who manifest no nutritional problems, contrary to the old concept that it followed childhood rickets. It may be recognized as early as the second month of life; more commonly the alert parent or pediatrician will detect the depression of the sternum in the infant of four to six months of age.

Pectus excavatum is a structural deformity that progresses in severity with body growth. It is caused by abnormal anatomic relationships between the central attachments of the diaphragm and the sternum.[1] In the anterior midline the diaphragm inserts on the xiphoid cartilage below or at its junction with the body of the sternum. When body growth progresses normally, the diaphragmatic attachment to the xiphoid is lengthened with the increasing anteroposterior diameter of the expanding thorax. If, for some reason, this attaching structure cannot lengthen adequately it will compromise the anterior growth of the xiphoid and the lower sternum. This may be visualized by considering the central attachment of the diaphragm as an unyielding, ligamentous guy rope between the soft, cartilaginous xiphoid and the vertebral column. When the thorax is contracted in expiration, the guy rope is slack and the deformity minimal or nonexistent; during full inspiration the diameter of the bony thorax increases and the guy rope becomes taut, restricting the anterior excursion of the xiphoid. In the infant this deformity, apparent only during inspiration, is first observed when he cries. As growth progresses the taut guy rope affects the full cycle of respiration and the xiphoid depression appears to deepen. Actually the bony thorax is growing away from the restricted lower end of the sternum (fig. 1).

There are secondary changes which appear as the pectus excavatum is established. The costal cartilages connecting the depression with the expanding rib cage are gradually "warped" into the depression. When the pectus excavatum causes severe depression, the anteroposterior shortening of the lower chest restricts the lower mediastinum. The result is a decided displacement of the heart and major vessels to the left of the midline. Whether severe mediastinal deviation associated with pectus excavatum is in itself clinically significant is debatable.

The cosmetic trauma of pectus excavatum in a child of school age and a young adult is significant.[2] The unsightly appearance of the sternal depression will inevitably arouse comments and curiosity from schoolmates and companions. These patients are reluctant to take part in athletic programs or other group activities where the chest deformity may be exposed. A feeling of physical

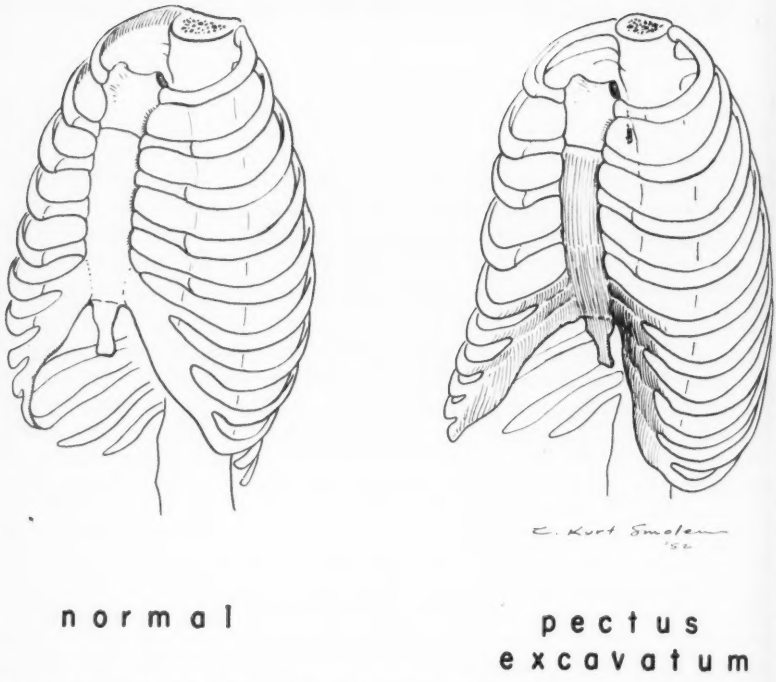

Fig. 1. A comparison of the normal bony thorax with that of pectus excavatum reveals the depression and associated costal flare. Only the anteroposterior diameter of the lower thorax is involved; compression and deviation of the mediastinum result.

inferiority and resultant personality impairment are frequently associated with pectus excavatum.

SURGICAL TREATMENT

There are two phases of corrective therapy in pectus excavatum: (**a**) *infantile*, and (**b**) *adult*. The treatment is surgical in both phases, but the extent of surgery required differs considerably.[3] Therefore, early recognition and prompt correction are especially advantageous to the patient and his parents.

(**a**) The *infantile form* of pectus excavatum is corrected with minimal effort and the element of risk is correspondingly low. The ideal age for surgical intervention is four months.[4] At this age the impending deformity may be recognized and evaluated; the rib cage is soft and there is no fixed disfigurement. The operation consists only of resecting the xiphoid cartilage and releasing any adjacent fibrous attachment between the diaphragm and the lower sternum.

Technic. The infant is kept in the supine position when open drop ether is administered. An incision is made in the midline, directly over the xiphoid depression, and carried down to the rectus sheath. The sheath of the rectus is divided in the midline, and the lower border of the xiphoid cartilage is grasped by hemostat and elevated. By dissection with scissors the xiphoid is cut away from all muscular and fibrous attachments; the junction between the xiphoid and the body of the sternum is identified and the cartilage transected at that level. The tip of the index finger is used to determine any restricting diaphragmatic attachments to the lower end of the sternum; usually there are none. One or two sutures are used to close the rectus sheath and several more employed for skin closure. The postoperative care is simple; only one or two days of hospitalization are necessary. The skin sutures may be removed in five days at the patient's home or in the office.

This simple operation releases the restricting diaphragmatic attachment to the lower sternum and xiphoid. Normal growth of the bony thorax is permitted, and unnecessary deformity of pectus excavatum is avoided.

(**b**) In the *adult form* of pectus excavatum the surgical correction taxes the ingenuity and patience of the surgeon. The fixed bony depression together with the flattened thorax and the flaring costal margins present a difficult problem. In many instances the deformity is so severe that anticipated improvement must be considered on a relative basis.

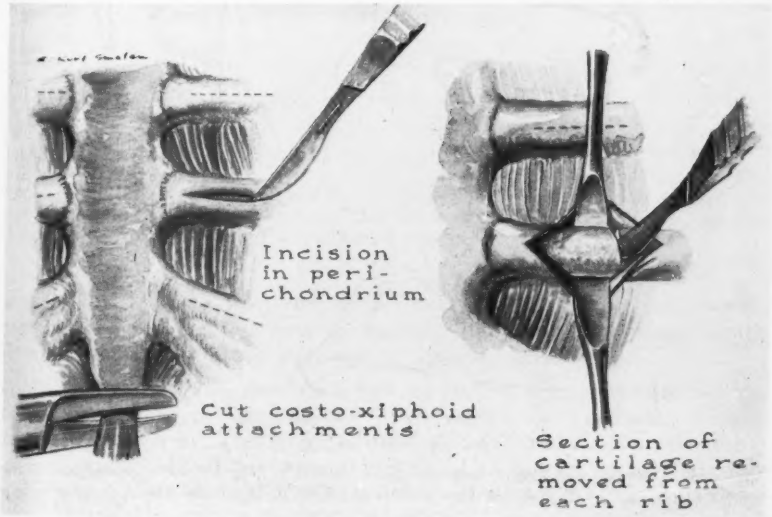

Fig. 2. After exposure through a midline incision the lower sternum is mobilized by partial resection of the costal cartilages and the xiphoid. Care is taken to preserve the perichondrium.

Technic. Endotracheal gas-oxygen anesthesia is employed. The patient is in a supine position. The incision is midline and extends the full length of the sternum. The skin edges are undermined, the plane of cleavage lying between subcutaneous fascia and pectoral muscles, from the midline laterally to the costochondral junctions. The pectoral muscles are incised directly over the third costal cartilages, and the perichondrium is cut along the long axis of the rib. The first and second cartilages are not disturbed. The perichondrium is carefully stripped from the cartilage and the cartilage excised from sternum to the bony rib. This process must be repeated for each of four to six pairs of cartilages (fig. 2). Emphasis is placed on the need for preserving the perichondrium, otherwise a soft chest wall will result on either side of the sternum. When properly preserved, the youthful perichondrium will regenerate cartilage.

Excision of eight or ten costal cartilages is a tedious surgical procedure. The surgeon is tempted to shorten the operation by breaking or transecting the

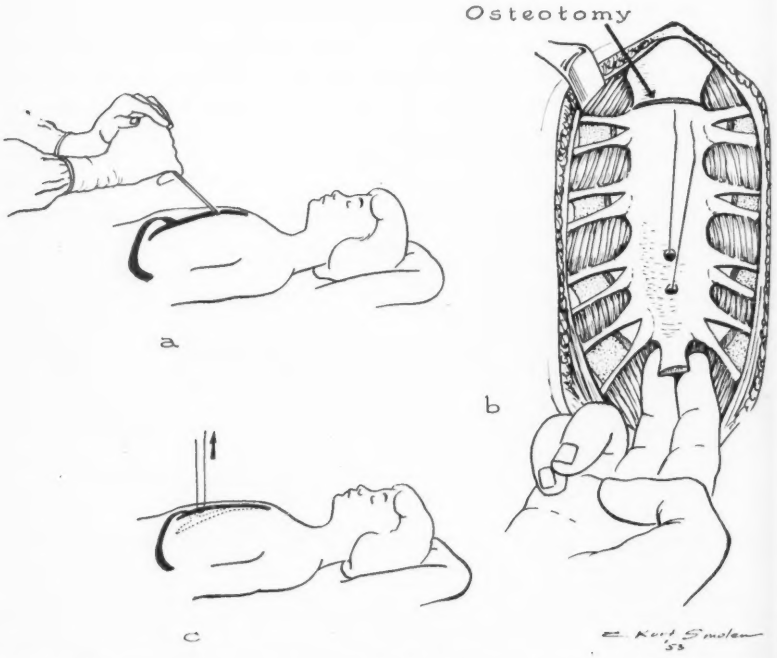

Fig. 3. (a) A wedge osteotomy is made in the anterior plate of the sternum below the manubrial junction. This may be done with bone chisel or bone saw. (b) A loop of wire is employed for continuous traction on the mobilized segment of sternum. Additional drill holes may be made if more wire loops are desired. (c) Elevation of the mobilized segment before closure of the wound indicates the degree of traction necessary. Usually rubber band traction is used to compensate for respiratory excursion.

Fig. 4. Sketch of the plaster jacket and simple countertraction bar. A window is placed over the operative area for care and inspection. The single wire loop in this case is suspended by an ordinary rubber band.

cartilages and literally fracturing the sternum into place. The best cosmetic result will be obtained by complete removal of the "warped" cartilage which permits regeneration of new cartilage with normal contour.

After excision of the costal cartilages the xiphoid is disarticulated and removed. The retrosternal space is easily entered and the lower end of the sternum elevated by retractor. The flimsy mediastinal fascia and loose areolar tissue are easily separated from the posterior aspect of the sternum by blunt dissection. Mobilization of the deformed sternum is carried up to the second interspace where the sternal body and manubrium articulate. The level where the depression begins is selected and a transverse wedge osteotomy is performed through the anterior plate of the bone. This may be done with a saw or chisel

(fig. 3a). The sternum is then elevated to proper position for correction of the deformity.

Fixation is necessary during the early period of cartilage regeneration. A simple method of wire traction is now employed (fig. 3b). Methods employing rib struts, grafts, and internal fixation should be utilized with caution. By the simple measure of a wire loop through two drill holes in the lower sternum, a system of constant, rubber band traction has been used (fig. 3c). A light plaster jacket is made before the operation and is bivalved. A window is cut over the operative site and a bar for countertraction incorporated in the cast (fig. 4). The light cast permits prompt ambulation of the patient and also affords protection to the chest wall. Traction is maintained for three or four weeks, and the wire loop is then removed. In active children the jacket may be worn as a protective vest for an additional period of time.

One detail that deserves special mention is the accurate evaluation of the defect before surgical correction is attempted. Fluid may be poured into the hollow defect while the patient is supine and the amount accurately measured. This is sometimes difficult in the very small child. Radiographic studies are valuable; of these, the true lateral projection in both expiration and inspiration will be most helpful. Accurate measurement of the deformity will assist in evaluating the operative procedure.

SUMMARY

Pectus excavatum is an acquired lesion and may occur in an otherwise healthy person. The deformity is primarily of cosmetic significance and to a lesser degree impairs health or well being. The degree of deformity is directly dependent upon the fixation of the terminal sternum and the xiphoid cartilage.

Correction of pectus excavatum is a surgical procedure and two methods of correction are described. Selection of the type of operation depends on the age of the patient and the degree of sternal fixation.

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THE IMPORTANCE OF FECAL EXAMINATION IN THE DIAGNOSIS OF STRONGYLOIDIASIS

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BETWEEN 1942 and 1952 there were 35 patients seen at the Clinic in whom diagnoses of infestation with *Strongyloides stercoralis* were made. This was the most commonly reported nematode, occurring slightly less than once in each thousand stools examined. This report summarizes the symptoms of which the infested patients complained, and emphasizes the importance of fecal examination in the diagnosis of this disease.

The life cycle of *S. stercoralis* is similar to that of the hookworm but is slightly more complicated. The filariform larvae develop in soil and infest the host by penetrating the skin, and eventually reach the intestines by way of the heart and lungs where they develop into adults. The adults mate, and the females penetrate the intestinal mucosa where the eggs are laid. These hatch into rhabditiform larvae which are released into the lumen of the intestines. Detection of the parasite depends upon observation of larvae in the feces. These forms are quite easy to identify because *S. stercoralis* is the only common parasite in which the ova hatch within the intestines. The larval form pass into the soil and the life cycle may then follow either of two courses. The rhabditiform larvae may develop directly into adults whose eggs hatch into more rhabditiform larvae which in turn can develop into adults. The second type of life cycle is the parasitic one in which the rhabditiform larvae develop into filariform larvae which have the power of infesting man. In man, furthermore, infestation is self-perpetuating because the larvae can penetrate the colonic mucosa or perianal skin to produce auto-infection.[1,2]

Since the original description of this parasite by Normand[3] in 1876, many workers have described the occurrence of these parasites in human disease and have discussed their role in the production of the symptoms observed. In general, the ability of the parasite to produce symptoms is recognized, although many patients who harbor the parasites have no complaints which can be related to this infestation.[4]

On the basis of the parasite's life cycle, these symptoms would be expected to be confined to the skin, lungs and gastrointestinal tract. The symptoms referable to the gastrointestinal tract are variable. The chief of these is abdominal pain which may occur from the epigastrium to the lower abdomen and may be diffuse or well-localized. It has been described as colicky, cramp-like, gnawing, dull, and heavy. Food has a variable effect on it. Some patients find that their discomfort is exacerbated by eating, others feel better afterwards, and still others believe that food has no effect on their condition. Diarrhea, frequently bloody, is said to be present, but others complain of constipation. Weight loss, nausea, vomiting, malaise, weakness, anorexia, jaundice, and

indigestion have been mentioned. Anemia, leukocytosis and eosinophilia are the only laboratory findings of significance aside from those in the stool examinations. Several reports have described strongyloidiasis of the urinary tract but this is of rare occurrence.[5] All of the clinical signs and symptoms associated with this disease are nonspecific for any of them may occur in patients with other gastrointestinal tract disease.

In this series of 35 patients, diagnoses were made through demonstration of the larvae in this laboratory in all but one case; in the majority of patients the final diagnoses would have been that of a functional disease had the parasites not been found. The one diagnosis, not confirmed by laboratory demonstration of the parasite here, was made on the basis of a history of the demonstration of the larvae at another hospital.

A stool examination is a simple procedure, requiring a little practice at recognizing the presence of parasites, a microscope, and containers for the feces specimens. In our laboratory, pint ice cream containers are used for collecting the specimens. It is a simple matter to make an emulsion of feces in a little saline on a slide. Best results are obtained from normally formed or soft, mushy stools, produced naturally or following a saline laxative. Examination of the stool specimen for 15 or 20 minutes reveals most parasites which occur as larval forms in strongyloidiasis. Of the 34 cases in the present series in which the parasites were found here, they were seen in the first stool specimen examined in all instances except one.

DESCRIPTION OF THE PATIENTS

Thirty-three of the 35 patients were adults; there were two children, both girls, two and a half and eleven years of age. Initial examinations of these patients were made by 22 different clinicians representing six departments. The significance of the sex distribution among the adults, 26 men and 7 women, is not immediately apparent although possibly men are subject to greater exposure than women. Occupations seemed to play no part in the epidemiology of the disease. Of the 35 patients, eight gave a history of having lived or traveled extensively outside of the United States. Three were veterans who had histories indicating previous parasitic infestations. Of the group in which the infestations were seemingly contracted in this country, eight were from the Cleveland-Akron area and six from other cities and villages in Ohio, 12 were from West Virginia and one from Pennsylvania.

CLINICAL CONSIDERATIONS

On admission 22 of the group mentioned gastrointestinal symptoms as their major or only complaint. Five patients were seen with skin conditions which might possibly be related to strongyloidiasis. Three patients were seen because of eosinophilia which proved to be caused by strongyloidiasis. The

others were admitted for symptoms entirely unrelated to their parasitic infestation. The chief complaints of the patients which were believed to have possible connections with the presence of the parasites are summarized in Table 1.

Table 1

"Chief Complaints" Believed to be Due to *Strongyloides stercoralis* Infestation in 35 Patients

Upper abdominal pain	11
Lower abdominal pain	7
Change in bowel habits	6
diarrhea 3	
constipation 1	
"change" 2	
Gas in stomach, rumbling in bowels, etc.	5
Urticaria, rash, peeling	5
Hematologic disorders (eosinophilia)	3
Nervousness	3
Unrelated complaints	16

After a review of the charts in an attempt to determine which, if any, symptoms or signs are of significance in the diagnosis of this disease, it became apparent that none is pathognomonic of the presence of the parasite with the exception of the detection of the larvae of the nematode in the stool of the patient. Abdominal pain which is usually mentioned first among such symptoms occurred in 71 per cent of this series and was so variable that any attempt to characterize it fails; the most common type recorded was lower abdominal pain, most frequently described as a heavy, dull pain which occasionally became colicky. Gas, bloating, meteorism, and excessive flatus are too easily considered neurotic symptoms and dismissed as evidence of the functional nature of a patient's illness. However, 88 per cent of the patients in this series suffered from some form of excessive gas production or accumulation; indeed, a number of them listed this as their only presenting complaint. Change in bowel habits is a common symptom described as associated with parasitism. In this series constipation was much more frequent than diarrhea, contrary to most of the textbook descriptions.[1] Blood in the stool is usually described in the literature but occurred in only 20 per cent of this series (Table 2).

Of the patients on whom differential blood counts were available, only two had white counts above 11,000. One of these was hospitalized because of eosinophilia as it had been noted at another hospital that her white count was 45,000 with 75 per cent eosinophils. By the time she was seen in our hospital, her white count had dropped to 15,450 with 45 per cent eosinophils. Further investigation led to the discovery of *S. stercoralis* larvae in her stool. The other patient with leukocytosis had a total white blood cell count of 19,000 with 8 per cent eosinophils, but clinically did not differ from other patients in the

series. Four of the patients had eosinphil counts of 4 per cent or less and were considered normal; the remainder ranged from 6 to 22 per cent. As there was no method of determining the duration of infestation, it was impossible to correlate it with the eosinophil count as suggested by Liebow and Hannum.[6] None of the patients showed anemia.

A review of some of the working impressions recorded following the initial physical examination shows that only 13 of these patients were suspected of having parasitism, and in many instances this was considered to be sufficiently improbable that the term "rule out parasites" was used. There were 43 initial tentative diagnoses referable to the gastrointestinal tract and the majority of these were of functional conditions. There were, however, five impressions which were strictly organic in nature, involving possible neoplasms or gallbladder disease. Ten were associated with skin disease which may or may not have any relationship to intestinal parasitism. One possible hematologic diagnosis, that is "rule out leukemia," proved to be related to the patient's parasitic infestation. The remainder of the impressions were directly related to definite pathologic conditions patients presented.

The final diagnoses, other than strongyloidiasis, entered in the charts covered almost every system of the body as might be expected in patients with such many and varied complaints. It is interesting that of the entire series, with the exception of the patient whose primary diagnosis was carcinoma, none of the patients were seriously ill. This contradicts the experience of many others who have noted that the patient with an infestation of this type can be extremely ill. Because the great majority of these patients are referred back to their own physicians for follow-up, the results of treatment cannot be evaluated here. In all cases the recommended treatment was gentian violet tablets.

Table 2

Most Common Signs and Symptoms Associated with *Strongyloides stercoralis* Infestation in 35 Patients

Parasites found in stool	97%
Accumulation of gas, belching, distension, flatus	88%
Abdominal pain	71%
epigastric	14%
lower	57%
Eosinophilia*	71%
Change in bowel habits—diarrhea, constipation	54%
Energy loss	28%
Blood in stool	20%
Foreign residence	20%
Other intestinal parasites present	17%
Skin conditions—urticaria, rash, peeling	14%
Indigestion	11%

* *Ten out of 14 patients only.*

SUMMARY

The diagnosis of infestation by the nematode *Strongyloides stercoralis* is primarily made on laboratory examination; aside from actual observation of larvae in the stool, there is no laboratory test that is diagnostic. In 33 out of 35 patients seen during a ten year period, there was no suggestion in the patients' admitting diagnoses of the possibility of parasitism with this organism. In one instance in which strongyloidiasis was suggested prior to fecal examination, diagnosis was made on the basis of a definite history of discovery of the larvae at another hospital. There are no physical signs pathognomonic of the disease. The history of gastrointestinal distress is so common in patients seen in departments of general medicine or gastroenterology that it offers little help in establishing a diagnosis. The importance of routine stool examinations for parasites in patients with any type of gastroenterologic distress cannot be overemphasized.

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THE TAARNHOJ* OPERATION:

RELIEF OF TRIGEMINAL NEURALGIA WITHOUT NUMBNESS

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TAARNHOJ[1] in 1952 reported ten consecutive patients with trigeminal neuralgia who were treated by dividing the dural sheath enclosing the sensory root of the gasserian ganglion without dividing the nerve itself. This procedure afforded these patients relief of their tic douloureux without resultant anesthesia of the face. The reasoning which led Taarnhoj to attempt this procedure was suggested by the following case.

In April 1951 Taarnhoj observed a 31 year old man with typical trigeminal neuralgia of the third division. Because of the patient's age and the location of his pain, he suspected that the neuralgia was caused by an epidermoid in the right cerebellopontine angle, as had been previously reported by Olivecrona.[2] At operation an epidermoid about the size of a hazelnut was found which was compressing the trigeminal root. The epidermoid was removed and the trigeminal root was left intact. When last seen seven months after operation, the patient had no pain and had normal sensation in the face.

This experience led Taarnhoj to suspect that idiopathic trigeminal neuralgia might also be due to compression of the root of the nerve. In studying the anatomy of the nerve it appeared to him that the most likely spot where compression might occur was in the narrow channel formed by the dura as it passes over the upper margin of the petrous bone. Therefore, he decided to treat patients with trigeminal neuralgia by dividing the dural sheath enclosing the root and ganglion without dividing the root itself. All of his patients had relief of their tic pain when last seen from one to eight months after the operation.

Love[3] reported a case of a patient treated by this method in which an extradural approach instead of the intradural method of Taarnhoj was used.

We were not eager to try this new form of treatment because we doubted that it would stand the test of time. Everyone who has treated tic douloureux has seen many forms of therapy enthusiastically advocated in a preliminary report only to find that the author advocating the new treatment has found his enthusiasm waning about the time the article appeared in print. However, in December 1952 a patient appeared and requested "the new operation for trigeminal neuralgia which did not produce numbness." Nine years before this admission, the patient had had a total resection of the left sensory root for tic douloureux. Four and a half years later he developed tic douloureux on the

* *Pronounced "Tarnoy"*

right side, and on this side a partial resection of the sensory root afforded relief. A partial instead of a complete rhizotomy was performed because the patient complained so bitterly of the left facial numbness which followed the first operation and of the impossibility of chewing food on this side. In July 1952 the tic recurred on the right side. He had by this time developed definite symptoms and signs of multiple sclerosis which was undoubtedly responsible for his tic douloureux. In spite of extremely severe pain this patient was still unwilling to accept numbness of the right side of the face in exchange for relief. The physician who was treating his multiple sclerosis suggested "the new operation which would not produce numbness," and the patient wished to have it done. With this stimulus it was decided to apply Taarnhoj's procedure in this case, and in a series of patients with idiopathic trigeminal neuralgia.

In the past two months this operation has been performed upon nine patients with trigeminal neuralgia. We were surprised to find that not one of these patients has experienced a single paroxysm of trigeminal neuralgia after recovering from the anesthetic of the operation. Some of these patients had areas of mild sensory impairment in the trigeminal distribution apparently due to the trauma of dissecting the dura from the ganglion sheath and sensory root. As a rule this sensory loss cleared within a few days following the operation. The first patient operated upon had a mild postoperative aphasia which persisted for several days. In this case the transdural technic advocated by Taarnhoj was employed. In the subsequent cases the extradural approach devised by Frazier was employed and the dura was not incised until the sensory root was exposed. This approach, advocated by Love adds to the safety of the operation. Incision of the dural sheath is a more difficult technic than the Frazier-Spiller operation of resection of the sensory root. Also, it entails some risk of injury to the fourth cranial nerve, but if the results are lasting, the technic will be perfected, the risks minimized, and the numbness and painful paresthesias which sometimes follow the Frazier-Spiller operation, will be avoided.

Why does this procedure relieve the pain of tic douloureux? We have not opened the dural sheath over the ganglion itself but merely over the sensory root, and there seems to be adequate room in the sleeve for the fibers of the root. Furthermore, patients with tic douloureux seldom show evidence of impairment of sensory function as would be expected if the root were being compressed; nor does their pain resemble the pain of sciatica produced by compression of a lumbar nerve root by a protruded intervertebral disk. Tic douloureux is limited to the fifth nerve except for an occasional occurrence in the distribution of the glossopharyngeal nerve, and an extremely rare occurrence in the sensory root of the facial and of the vagus nerves.

The relief of tic douloureux by Taarnhoj's operation offers fascinating grounds for speculation regarding the etiology of this unusual type of pain. We would like to advance the theory that the pain of tic douloureux is due to the development of an artificial synapse in the sensory root fibers where the nerve crosses the apex of the petrous bone. This artificial synapse occurs as

the result of the demyelinizing processes and the development of sagging of the tentorium which accompany advancing age. Tentorial sag undoubtedly occurs with age as a result of the upright posture which man has adopted. The sagging of the tentorium where it merges with the roof of the dural sheath transforms the normal oval-shaped dural foramen which transmits the nerve into a relatively flat slit. This may be accentuated also by mild platybasia due to the osteoporosis which frequently occurs with the aging process. With this change in the shape of the dural foramen, the filaments of the sensory root, normally dispersed in the arachnoid sheath distended with cerebrospinal fluid, are held in contact with one another. Contact of the various nerve elements in the sensory root, together with demyelinization of some of the axis cylinders, permit short circuiting of the action current which accompanies the transmission of the nervous impulse, with the formation of an artificial synapse.

Two articles are especially pertinent to the elaboration of this theory. In 1944 Granit, Leksell and Skoglund[4] showed that an artificial synapse is produced in a mixed nerve by injury or pressure on the nerve. They found that this pressure may be so mild as not to produce interference with normal nerve transmission, and yet due to loss of the insulating myelin sheath, a portion of the impulse may pass from an efferent to an afferent fiber. For instance, in causalgia they believe that the efferent sympathetic impulse reaches the point of compression of the nerve and jumps across to the naked axis cylinder of the pain fiber. Thus the efferent sympathetic impulse is converted into an afferent pain impulse. According to Lewy, Groff, and Grant,[5] the trigeminal nerve in the cat, in addition to its well known somatic afferent fibers and the efferent fibers to the muscles of mastication, contains also some autonomic efferents which are cholinergic in character and therefore, probably parasympathetic. These supply muscle fibers in the tongue, the cat's whiskers, the upper lip, and the elevator of the eyelid. These two contributions together with the fact that trigeminal neuralgia is relieved by Taarnhoj's operation, suggest the following theory to explain the mechanism of tic douloureux.

A tactile stimulus originating in the trigger zone, commonly located in the whisker area, is carried in the somatic afferent fiber to the brain stem. Here it forms a reflex connection with autonomic cells which Lewy, Groff, and Grant, have shown are probably in the mesencephalic nucleus of the fifth nerve. This results in an efferent impulse which travels in the autonomic fibers accompanying the sensory root. When it reaches the artificial synapse at the point of compression of the nerve root, this impulse is short circuited into the naked pain fiber, and is reflected back into the brain stem as a painful impulse.

Thus we believe that tic douloureux, like causalgia, is the result of cross circuiting of autonomic impulses into somatic pain fibers. Tic douloureux is due to a preganglionic parasympathetic-sensory synapse while causalgia is due to a postganglionic sympathetic-sensory synapse. The difference in the character of the two types of pain may be explained by the fact that the sympathetic discharge is diffuse and prolonged while the parasympathetic discharge is discrete and of brief duration.

This theory explains beautifully the mechanism of the trigger action, and also why the pain of tic douloureux may be relieved by blocking the nerve peripheral to the ganglion even though the site of the lesion is proximal to the ganglion. It explains why the whisker area is so commonly a trigger zone. It explains why the pain of tic douloureux is limited to the fifth and ninth nerves. It explains why the paroxysms of glossopharyngeal neuralgia are more apt to be precipitated by tart flavors or even, in some instances, by merely thinking of them. It explains the association of tic douloureux with multiple sclerosis and why it occurs most frequently in the elderly. It explains how tic douloureux can be relieved by Taarnhoj's operation.

Fortunately, Taarnhoj is a young man. If he were as old as the senior author of this article, he would have realized that his idea couldn't possibly work and he wouldn't have tried it.

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Those wishing to attend should fill in the application form on page 371 and return it as soon as possible.

TENTATIVE PROGRAM

Wednesday, May 13, 1953

Morning Session

F. A. LeFevre, M.D., Presiding

8:00-9:00 a.m.	Registration	
9:00-9:10 a.m.	Opening Remarks	Herbert Salter, M.D.
9:10-9:30 a.m.	Indications for Cholecystectomy	R. S. Dinsmore, M.D.
9:30-9:50 a.m.	Treatment of Duodenal Ulcer	George Crile, Jr., M.D.
9:50-10:10 a.m.	Appraisal of the Cardiac Risk in Abdominal Surgery	A. C. Ernstene, M.D.
10:10-10:30 a.m.	Anesthesia in Surgery of the Abdomen	D. E. Hale, M.D.
10:30-10:50 a.m.	Intermission	
10:50-11:10 a.m.	Common Superficial Fungus Infections	G. H. Curtis, M.D.
11:10-11:30 a.m.	Use and Abuse of Cortisone and ACTH in Dermatology	J. R. Haserick, M.D.
11:30 a.m.-12:15 p.m.	Panel—Diseases of the Colon	R. S. Dinsmore, M.D. George Crile, Jr., M.D. R. B. Turnbull, Jr., M.D. E. N. Collins, M.D. J. C. Root, M.D.
12:30 p.m.	Luncheon—Courtesy Bunts Institute	

Afternoon Session

1:30-2:00 p.m.	Treatment of Coronary Artery Disease	A. C. Ernstene, M.D.
2:00-2:20 p.m.	Management of Varicose Veins and the Stasis Syndrome	A. H. Robnett, M.D.
2:20-2:40 p.m.	Clinical Significance of Hematuria	E. F. Poutasse, M.D.
2:40-3:00 p.m.	Management of Cancer of the Rectum	R. B. Turnbull, Jr., M.D.
3:00-3:30 p.m.	Results of Surgery for Cancer of the Stomach	C. H. Brown, M.D.
3:30-3:40 p.m.	Intermission	

3:40-4:10 p.m.	The Irritable Colon	E. N. Collins, M.D.
4:10-5:00 p.m.	Panel—The X-Ray in Diagnosis of Abdominal Surgical Disorders	S. O. Hoerr, M.D. C. R. Hughes, M.D. H. R. Rossmiller, M.D. George Crile, Jr., M.D.

Thursday, May 14, 1953

Morning Session

9:00-9:20 a.m.	Management of Surgical Patients with Diabetes	R. W. Schneider, M.D.
9:20-9:50 a.m.	The Action and Use of Antithyroid Drugs and Radioactive Iodine	J. R. Cook, M.D.
9:50-10:20 a.m.	Acne Vulgaris and Rosacea	G. H. Curtis, M.D.
10:20-10:50 a.m.	Headache	L. L. Lovshin, M.D.
10:50-11:10 a.m.	Intermission	
11:10-11:30 a.m.	Hypothyroidism and Myxedema	P. G. Skillern, M.D.
11:30-12:00 noon	Recent Advances in Treatment of Hypertension	I. H. Page, M.D.

REGISTRATION BLANK

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